No. 23-16026

IN THE UNITED STATES COURT OF APPEALS
FOR THE NINTH CIRCUIT

HELEN DOE, parent and next friend of Jane Doe; et al.,

Plaintiffs-Appellees,

v.

THOMAS C. HORNE, in his official capacity as State Superintendent of Public Instruction; et al.,

Defendants-Appellants,

and

WARREN PETERSEN, Senator, President of the Arizona State Senate; BEN TOMA, Representative, Speaker of the Arizona House of Representatives,

Intervenor-Defendants-Appellants.

On Appeal from the United States District Court
for the District of Arizona

EXHIBITS TO INTERVENOR-DEFENDANTS-APPELLANTS' EMERGENCY MOTION UNDER CIRCUIT RULE 27-3 FOR A STAY PENDING APPEAL

VOLUME 2

Exhibit 22

https://theconversation.com/when-it-comes-to-sport-boys-play-like-a-girl-80328

Marnee McKay

9 min read

When it comes to sport, boys 'play like a girl'

Primary school-aged boys and girls can play in mixed teams until they reach high school, our research suggests. Clappstar/Flickr, CC BY-SA

Published: August 3, 2017 4.16pm EDT

Authors

1. [Marnee McKay](#)

   Lecturer of Musculoskeletal Physiotherapy, University of Sydney

2. [Joshua Burns](#)

   Professor, Sydney Children's Hospitals Network, University of Sydney

Disclosure statement

Joshua Burns receives funding from NIH (National Institute of Neurological Diseases and Stroke and National Center for Advancing Translational Sciences, Inherited Neuropathies Consortium, Rare Disease Clinical Research Network #2U54NS065712), Charcot-Marie Tooth Association of Australia, Charcot-Marie Tooth Association (USA), Diabetes Australia, Multiple Sclerosis Research Australia, Sydney Southeast Asia Centre, New Zealand Neuromuscular Research Foundation Trust, Elizabeth Lottie May Rosenthal Bone Bequest and Perpetual Limited.

Marnee McKay does not work for, consult, own shares in or receive funding from any company or organization that would benefit from this article, and has disclosed no relevant

affiliations beyond their academic appointment.

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Girls in primary school are just as physically capable as their male classmates, according to our research, taking the sting out of the insult “you play like a girl”.

When we compared primary school children’s physical capabilities, differences between girls and boys were not as important as people think. So, they should be happily playing with and competing against each other in the backyard, playground and sporting fields.

Read more: ***It's not just the toy aisles that teach children about gender stereotypes***

As part of wider research to assess people’s physical capabilities across the lifespan, we tested 300 children and adolescents between the ages of 3 and 19.

We tested each child for over two hours, taking more than 100 measurements. These included measuring the strength of 14 muscle groups, the flexibility of 13 joints and 10 different types of balance. We looked at factors including hand dexterity, reaction times, how far kids could walk, how high and how long they could jump, as well as their gait.

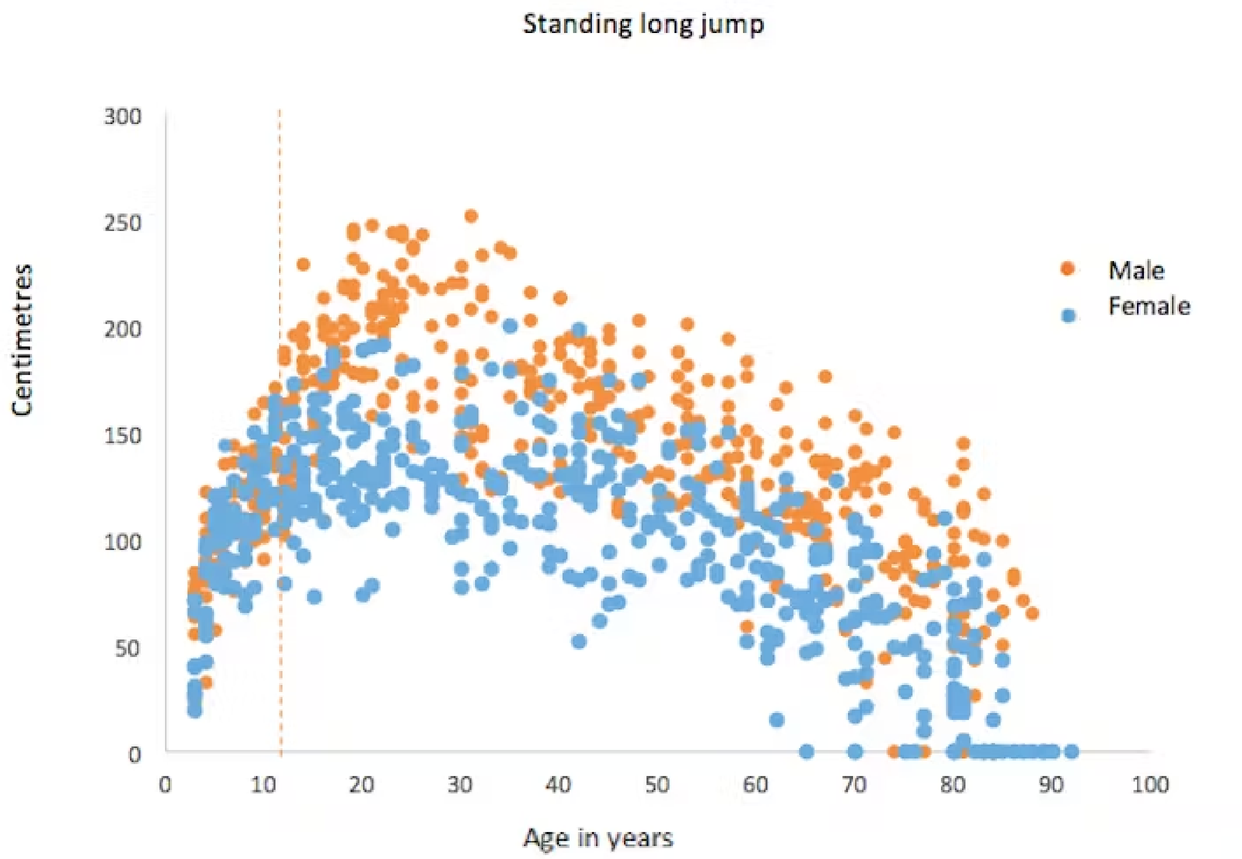
What did we find?

Across all measures of physical performance, there was one consistent finding. There was no statistical difference in the capabilities of girls and boys until high-school age (commonly age 12).

Let's use standing long jump (also known as a broad jump test) as an example. This provides a measure of your legs' explosive power. It needs minimal equipment and the results compare well with the type of information you get from strength testing using expensive equipment. It's also one of the tests would-be American NFL (National Football League) players take to impress talent scouts.

The standing long jump is a test football scouts use to assess explosive power.

We found no difference between boys and girls before they turn 12 (see graph below). Every physical measure followed this pattern.



Before the age of 12, boys and girls do just as well as each other in the standing long jump. Author provided

How do our findings compare?

Other studies have had similar results. These have included ones testing muscle strength, walking, jumping and balancing.

However, it's difficult to directly compare data from one study to another, as different studies have different sample sizes, include children of different age ranges, and assess different measures. For example, we were the first to use the timed stairs test and stepping reaction time to find what regular children were capable of.

Some studies found differences in physical capabilities between primary school-aged boys and girls using the same types of tests we used. And others reported small differences in the jump height of boys and girls aged 6-17 years but not with the long jump.

These differences can in part be attributed to sampling methods that were limited to specific age ranges or locations and socioeconomic backgrounds, the latter potentially having a significant impact on physical health and activity.

By contrast, the children in our research were generally representative of the Australian population, using data from the Australian Bureau of Statistics about socioeconomic status, ethnicity and body mass index.

What do our findings mean for kids, coaches and parents?

There is no consensus across schools or among different sports about mixed-gender sports for primary school children.

For instance, boys and girls compete separately in most local Little Athletics after age five but field hockey can have mixed gender teams until age 17.

And in tennis, primary school-aged girls and boys play separately in singles matches but can play against each other in mixed doubles.

Our findings support the push for boys and girls to compete in mixed sporting teams until the end of primary school, after which the hormonal changes of puberty mean boys tend to perform better in sports and tasks requiring strength and speed.

Read more: ***Our 'sporting nation' is a myth, so how do we get youngsters back on the field?***

There are also some practical advantages to mixed sport in primary school and in weekend competitions:

- fewer scheduling conflicts for councils (allowing school and sport administrations to fit games more conveniently into busy sporting venues)

- fewer clubs or organisations to share already stretched government and private sector funding
- consolidation of coaching and manager talent, and most importantly
- fewer parent-taxi drop offs.

Perhaps perceived differences in physical capability between boys and girls are based on outdated gender stereotypes that appear at birth, when some boys are given their first footy and some girls their first doll.

But whatever the origin of the idea young boys are physically more capable than young girls, the evidence is clear. Boys “play like a girl”, and that’s certainly no insult.

Exhibit 23

Normative reference values for strength and flexibility of 1,000 children and adults

Marnee J. McKay, MHlthSc
Jennifer N. Baldwin, BAppSc (Hons)
Paulo Ferreira, PhD
Milena Simic, PhD
Natalie Vanicek, PhD
Joshua Burns, PhD
For the 1000 Norms Project Consortium

Correspondence to M.J. McKay: marnee.mckay@sydney.edu.au

ABSTRACT

Objective: To establish reference values for isometric strength of 12 muscle groups and flexibility of 13 joint movements in 1,000 children and adults and investigate the influence of demographic and anthropometric factors.

Methods: A standardized reliable protocol of hand-held and fixed dynamometry for isometric strength of ankle, knee, hip, elbow, and shoulder musculature as well as goniometry for flexibility of the ankle, knee, hip, elbow, shoulder, and cervical spine was performed in an observational study investigating 1,000 healthy male and female participants aged 3–101 years. Correlation and multiple regression analyses were performed to identify factors independently associated with strength and flexibility of children, adolescents, adults, and older adults.

Results: Normative reference values of 25 strength and flexibility measures were generated. Strong linear correlations between age and strength were identified in the first 2 decades of life. Muscle strength significantly decreased with age in older adults. Regression modeling identified increasing height as the most significant predictor of strength in children, higher body mass in adolescents, and male sex in adults and older adults. Joint flexibility gradually decreased with age, with little sex difference. Waist circumference was a significant predictor of variability in joint flexibility in adolescents, adults, and older adults.

Conclusions: Reference values and associated age- and sex-stratified *z* scores generated from this study can be used to determine the presence and extent of impairments associated with neuromuscular and other neurologic disorders, monitor disease progression over time in natural history studies, and evaluate the effect of new treatments in clinical trials. ***Neurology*®** **2017;88:36–43**

Meaningful, reliable, and sensitive outcome measures are required to monitor treatment and progression of neuromuscular and other neurologic disorders. While there have been substantial advances in the understanding of the pathogenesis and natural history of many neuromuscular disorders, the identification and development of new outcome measures that best reflect the efficacy of specific treatments have not advanced at the same rate.[1] Establishing valid and responsive outcome measures is a priority for the field.[2] To assist in the development of new outcome measures, normative reference values generated from large populations across the lifespan using standardized methods are required. Normative reference values can be utilized to generate *z* scores, which can be used in multicenter studies to improve outcome measure precision and responsiveness.

Muscle weakness and joint contractures predispose to numerous pathologies requiring intervention. Reference data play an important role in identifying and quantifying these impairments and evaluating the effectiveness of interventions. Currently, few comprehensive datasets detail the normal variation of active range of motion in healthy individuals and are limited by the number of joints assessed,[3] the age range of participants,[4,5] or insufficient sex representation.[6,7]

Supplemental data at Neurology.org

From the Faculty of Health Sciences (M.J.M., J.N.B., P.F., M.S., J.B.), University of Sydney, New South Wales, Australia; Department of Sport, Health and Exercise Science (N.V.), University of Hull, UK; and Sydney Children's Hospitals Network (Randwick and Westmead) and Paediatric Gait Analysis Service of New South Wales (J.B.), Children's Hospital at Westmead, Sydney, Australia.

Coinvestigators are listed at Neurology.org.

Go to Neurology.org for full disclosures. Funding information and disclosures deemed relevant by the authors, if any, are provided at the end of the article.

Similar limitations exist in strength reference datasets, relevant only to children[8–11] or adult populations,[12–16] or strength measured using equipment not readily available in clinic.[17–19]

The purpose of this study was to generate a reference dataset of normative values across the lifespan for an extensive set of isometric muscle strength and joint flexibility items, stratified for age and sex, and to investigate the influence of demographic and anthropometric factors.

METHODS

Study design and participants. Data were collected as part of the 1,000 Norms Project, an observational study investigating physical function and self-reported health in 1,000 people across the lifespan (see full protocol[20]). One thousand people aged between 3 and 101 years from the Greater Sydney metropolitan area in Australia participated in the project. Participants were recruited from January 2014 to September 2015 using highly structured convenience and snowball sampling techniques, including advertising via social media, e-newsletters, and community flyers. Presentations were held at social and volunteer groups, aged care organizations, playgroups, and schools. Eligible participants were aged ≥3 years, considered themselves healthy for their age, and could participate in age-appropriate activities of daily living. People with significant health conditions affecting physical performance or an inability to follow age-appropriate instructions were excluded. Potential participants with the following conditions were also excluded: diagnosed diabetes mellitus; malignant cancers; demyelinating, inflammatory, or degenerative neurologic conditions; pregnancy; class 3 obesity; severe cardiac or pulmonary disease; joint replacement; infectious or inflammatory arthropathies; or severe mobility impairment necessitating dependence on mobility aids for all ambulation. Equal numbers of male and female participants were recruited and were stratified into 9 age categories. One hundred people per decade were recruited in the age groups of 20–29, 30–39, 40–49, 50–59, 60–69, 70–79, and 80+ years. In order to represent the rapid periods of growth and maturation and to distinguish between young children and adolescents, 20 children per year from 3 to 9 years of age and 16 per year from 10 to 19 years of age were recruited.

Standard protocol approvals, registrations, and patient consents. Ethical approval was granted by the institutional Human Research Ethics Committee (HREC 2013/640) and written informed consent was obtained from all participants or parents/guardians of children.

Procedure. Participants attended the University of Sydney Performance Laboratory once for a 2-hour assessment. Participants had their height, body mass, waist circumference, and lower limb alignment measured. Foot structure was assessed using the Foot Posture Index, a 6-item summed scale from −12 (supinated) to +12 (pronated).[21] Age, sex, current work status, and self-reported ethnicity were collected from all participants or parents/guardians. Work status was classified as working (full-time, part-time, or unpaid) or not currently working (unemployed, student, or retired). Ethnicity was classified into 5 categories: British/European, American, Asian, African, and Aboriginal/Torres Strait Islander.

Two experienced clinical evaluators (physiotherapists) assessed isometric strength and joint flexibility using standardized methodology, including instructions, positioning, and scoring.[20] The dominant limb was assessed and determined as the hand used to write with and the foot used to kick a ball. The strength of 12 muscle groups—hand grip, ankle dorsiflexors and plantarflexors, knee flexors and extensors, hip abductors, internal and external rotators, elbow flexors and extensors, and shoulder internal and external rotators—were assessed by maximal voluntary isometric contraction using a portable hand-held dynamometer (Citec dynamometer CT 3001; CIT Technics, Groningen, Netherlands). The dynamometer was calibrated 0–500 N with certified weights monthly throughout data collection. The strength of knee musculature in participants ≥12 years of age was assessed by fixed dynamometry (CSMi; HUMAC NORM, Stoughton, MA). For unit of measure consistency, knee flexor and extensor strength in children aged 3–11 years were converted to Newton-meters (Nm) using anthropometric tables.[22] Rather than using the fixation device outlined in the protocol, ankle plantarflexion strength was assessed using hand-held dynamometry in long sitting, heel over plinth edge.

Joint flexibility was assessed using a universal goniometer, digital inclinometer, or bubble inclinometer (Baseline; Fabrication Enterprises Inc., White Plains, NY) depending on the joint assessed. Thirteen active joint range movements were assessed: ankle dorsiflexion and plantarflexion, knee flexion and extension, hip flexion, internal and external rotation, elbow flexion and extension, shoulder internal and external rotation, and cervical flexion and extension. Interrater reliability of the clinical evaluators demonstrated satisfactory repeatability of all strength and flexibility measures (intraclass correlation coefficient$_{2,1}$ 0.80–0.99) in a pilot study of 10 participants aged 6–67 years.

Data analysis. Data were collected and managed using REDCap electronic data capture and manually checked for transcription errors. Reference values were generated for each age group and sex in SPSS v22 Statistics for Windows (IBM SPSS; Armonk, NY). Normality of the data was assessed using Kolmogorov-Smirnov test. For analysis, age categories were children (3–9 years), adolescents (10–19 years), adults (20–59 years), and older adults (60+ years). To determine if strength and flexibility differed between male and female participants, independent t tests were conducted. A series of multiple regression models was constructed to determine the extent to which muscle strength and joint flexibility were influenced by participant demographic (age and sex) and anthropometric factors (height, body mass, waist circumference, foot posture, and lower limb alignment). First, Pearson product-moment correlation coefficients (r) were generated to explore the bivariate relationships between strength and anthropometric and demographic factors. The same correlations were explored for each joint flexibility measure. Second, factors identified to have an association ($r \geq 0.3$, $p < 0.05$) with strength or joint flexibility were entered simultaneously into a stepwise multiple regression model, which was reduced to a set of factors that best predicted and could be regarded as independent determinants of each strength and joint flexibility measure. To avoid multicollinearity, only one variable from highly correlated ($r \geq 0.7$) variables was included. Standardized β weights were calculated to provide an indication of the relative importance of the contribution of the various factors entered into the model to explain the variance in joint flexibility

Table 1 Isometric strength reference values of children (3–9 years), adolescents (10–19 years), adults (20–59 years), and older adults (60+ years)[a]

Muscle group	Entire sample	3–9 years		10–19 years		20–59 years		60+ years	
		Male	Female	Male	Female	Male	Female	Male	Female
Grip, N	187.1 (94.9)	55.8 (29.1)	50.6 (25.3)	195.7 (83.0)[b]	153.8 (46.0)	305.0 (73.0)[b]	190.2 (50.4)	221.6 (49.5)[b]	128.7 (35.4)
Ankle dorsiflexors, N	164.7 (61.2)	87.1 (38.2)	81.6 (29.2)	197.2 (56.6)[b]	166.0 (37.8)	224.6 (48.9)[b]	166.5 (41.6)	173.3 (44.0)[b]	131.5 (38.9)
Ankle plantarflexors, N	257.0 (85.6)	151.7 (52.3)	142.7 (45.9)	309.9 (74.9)[b]	261.2 (52.7)	338.8 (66.8)[b]	243.9 (59.2)	281.4 (62.7)[b]	216.3 (60.3)
Knee flexors, Nm[c]	68.6 (33.8)	27.0 (13.9)	25.2 (11.5)	89.8 (34.6)[b]	65.9 (19.7)	106.3 (28.6)[b]	64.4 (18.9)	76.3 (20.1)[b]	45.8 (13.3)
Knee extensors, Nm[d]	124.0 (66.4)	34.9 (18.1)	34.2 (14.9)	152.8 (71.1)[b]	116.9 (36.6)	202.1 (56.1)[b]	122.6 (33.6)	136.2 (35.6)[b]	81.9 (26.8)
Hip internal rotators, N	146.8 (69.5)	63.3 (31.7)	61.1 (25.8)	178.5 (67.2)[b]	143.2 (45.0)	217.7 (62.4)[b]	136.1 (44.6)	169.7 (55.0)[b]	108.4 (33.8)
Hip external rotators, N	110.4 (52.6)	49.7 (22.7)	43.8 (16.8)	141.7 (53.7)[b]	104.0 (28.7)	169.4 (45.8)[b]	100.7 (29.1)	125.5 (33.9)[b]	76.3 (23.7)
Hip abductors, N	116.1 (50.6)	52.3 (23.1)	52.4 (21.9)	143.4 (47.2)[b]	116.6 (31.9)	170.7 (43.9)[b]	113.1 (32.4)	124.8 (32.8)[b]	83.8 (23.5)
Elbow flexors, N	176.3 (80.0)	71.7 (29.1)	66.0 (26.4)	213.8 (81.1)[b]	148.5 (36.8)	270.2 (59.6)[b]	164.4 (42.3)	209.4 (48.4)[b]	129.7 (33.9)
Elbow extensors, N	135.8 (57.4)	66.8 (24.4)	62.0 (19.7)	159.3 (56.8)[b]	118.3 (30.0)	203.2 (46.1)[b]	121.2 (30.2)	162.1 (36.8)[b]	102.8 (25.3)
Shoulder internal rotators, N	126.7 (64.7)	56.1 (27.1)[e]	47.7 (17.4)	151.5 (63.2)[b]	101.6 (27.7)	202.4 (55.9)[b]	109.7 (33.6)	159.7 (42.9)[b]	86.0 (27.5)
Shoulder external rotators, N	86.4 (41.0)	38.7 (19.5)	34.7 (13.0)	100.6 (38.8)[b]	73.4 (19.1)	134.7 (39.6)[b]	82.2 (20.9)	96.7 (25.3)[b]	63.3 (19.2)

[a] Mean values (SD).
[b] Significant ($p < 0.01$).
[c] Participants aged 3–11 years measured with hand-held dynamometry (mean 94.4, SD 40.6) was converted to Nm.
[d] Participants aged 3–11 years measured with -held dynamometry (mean 126.4, SD 53.5) was converted to Nm.
[e] Significant sex differences ($p < 05$).

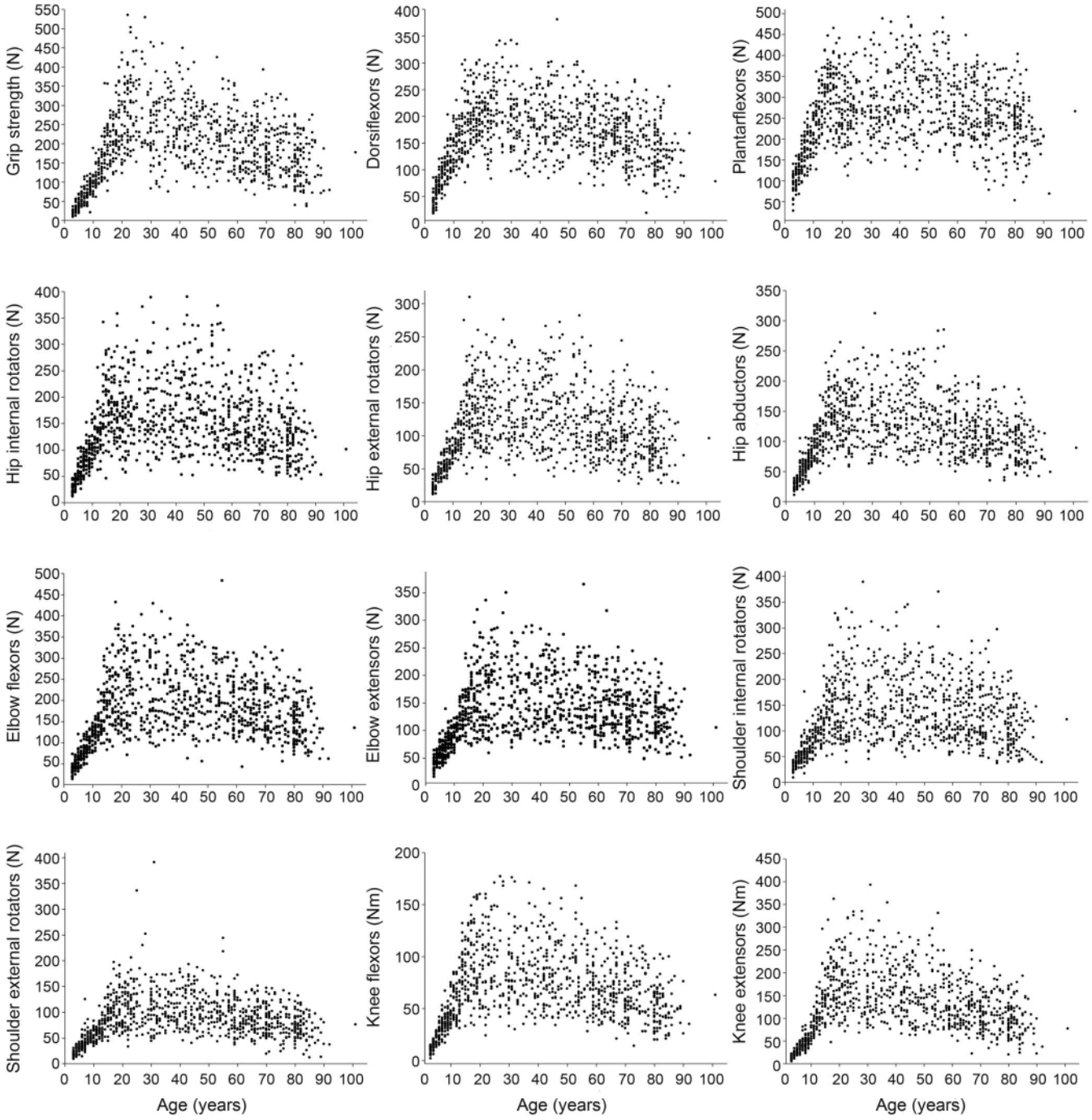
or muscle strength. Variables were retained in the multiple regression model if $p < 0.05$.

RESULTS To recruit 1,000 participants, 2,972 e-mails and 240 phone calls were logged. Ninety-one potential participants were excluded in accordance with the inclusion and exclusion criteria. Among adults aged over 18 years, 56% were currently working and 44% were not (31% retired and 13% students or unemployed). Participants were of diverse geographic ancestry, although the majority of participants were British/European ethnicity (74.4%), followed by Asian (16.6%), North or South American (5.1%), African (2.4%), and Aboriginal or Torres Strait Islander (1.5%). The sample mean (SD) age was 40.9 years (26.1), body mass 62.9 kg (21.1), height 1.61 m (0.02), waist circumference 78.6 cm (15.4), Foot Posture Index 3.5 (2.4), and lower limb alignment 1.8° (2.7). Ninety-three percent were right-footed and 91% were right-handed.

All missing data were accounted for. Four children declined to perform ankle dorsiflexion strength and 12 children were unable to perform cervical flexion and extension joint movements in accordance with the protocol. Ankle plantarflexors for 7 male adults and ankle dorsiflexors for 1 male adult were not assessed with hand-held dynamometry during periods of offsite calibration and servicing; 6 adults and 8 older adults were not assessed using fixed dynamometry due to safety concerns.

Normative reference values for the strength of 13 muscle groups per age category (children, adolescents, adults, and older adults) and sex are presented in table 1 and per decade in table e-1 at Neurology.org. From adolescence, male participants were significantly stronger in all muscle groups across all ages. There were no significant ($p < 0.05$) differences between the strength measures of boys or girls aged 3–9 years, except for shoulder internal rotators ($p = 0.031$), where boys were stronger. Correlations between strength and participant demographics and anthropometrics for children, adolescents, adults, and older adults are presented in table e-2. In children and adolescents, strength and age were highly correlated ($p < 0.05$), confirming that children become significantly stronger as they age from childhood and through adolescence. From 20 years of age, the relationship between strength and age changed. In adults aged 20–59 years, reduced strength with age was evidenced by significant, although weak, correlations with hand grip, ankle dorsiflexors, knee flexors and extensors, and shoulder external rotators. In older adults, decreased strength with increasing age was evidenced in all muscle groups. All muscle groups across all age categories demonstrated that greater height, body mass, and waist circumference were significantly associated with greater strength. The changes in strength measures with advancing age are shown in figure 1. Table e-3 shows the results of the multiple analyses. In children, height, followed

Figure 1 Scatterplots of muscle strength vs age for 1,000 children and adults



by waist circumference, was the most significant predictor of strength. In adolescents, a combination of body mass, sex, and age were shown to be the strongest independent predictors of strength. Sex (male) was the most significant predictor of strength in adults, followed by height and body mass with lower predictive values. In older adults, sex (male) was the most significant predictor, with body mass, height, and age demonstrating lower predictive values.

Normative reference values for active range of motion per age category (children, adolescents, adults, and older adults) and sex are presented in table 2 and per decade in table e-4. There was no significant difference ($p < 0.05$) in joint flexibility between boys and girls aged 3–9 years, except for hip internal rotation ($p = 0.017$), where girls had greater flexibility. Active range of motion was greatest in children compared to older adults. Figure 2 illustrates the inverse relationship between joint flexibility in all joints with age. Pearson correlations (table e-2) demonstrate that a decrease in flexibility with aging occurred in 8 of 13 joints of both adolescents and

Table 2 Joint flexibility reference values of children (3–9 years), adolescents (10–19 years), adults (20–59 years), and older adults (60+ years)[a]

		3–9 years		10–19 years		20–59 years		60+ years	
Movement, degrees	Entire sample	Male	Female	Male	Female	Male	Female	Male	Female
Ankle dorsiflexion	30 (6.7)[b]	33 (7.2)	31 (5.7)	32 (5.7)	31 (7.1)	32 (6.1)[b]	29 (6.4)	31 (6.1)[b]	26 (6.3)
Ankle plantarflexion	59 (8.6)[b]	63 (7.3)	63 (9.2)	58 (7.6)[b]	63 (7.3)	56 (7.5)[b]	62 (8.9)	53 (6.8)[b]	57 (7.2)
Knee flexion	137 (7.8)	145 (5.5)	144 (5.7)	140 (6.7)[c]	142 (6.6)	136 (6.1)	137 (6.2)	133 (7.2)	131 (8.1)
Knee extension	1 (2.9)	4 (3.3)	4 (3.9)	2 (2.6)	2 (2.6)	1 (2.3)	2 (2.7)	−1 (2.4)[c]	1 (1.6)
Hip flexion	121 (11.8)[c]	133 (9.1)	133 (9.8)	120 (9.9)[b]	124 (10.2)	120 (8.7)[b]	123 (10.0)	115 (10.7)	114 (12.6)
Hip internal rotation	37 (9.0)[b]	40 (8.4)	43 (9.1)	37 (9.3)	39 (7.7)	36 (7.9)[b]	40 (8.8)	33 (8.0)[c]	35 (8.4)
Hip external rotation	28 (8.5)[b]	32 (8.1)	32 (9.2)	31 (6.4)	31 (9.1)	30 (8.3)[b]	27 (8.3)	26 (7.0)[b]	22 (6.7)
Elbow flexion	148 (5.4)[b]	146 (5.4)	147 (5.8)	148 (5.4)[c]	150 (4.5)	147 (4.9)[b]	149 (5.4)	146 (6.0)[b]	149 (4.7)
Elbow extension	3 (5.9)[b]	7 (4.6)	7 (5.1)	4 (5.4)[b]	7 (5.6)	2 (5.0)[b]	4 (5.1)	−1 (5.0)[c]	0 (5.1)
Shoulder internal rotation	62 (12.9)[b]	67 (14.2)	67 (13.2)	62 (12.2)	66 (12.3)	58 (12.0)[b]	63 (14.0)	57 (11.0)[b]	63 (11.6)
Shoulder external rotation	83 (16.6)	98 (12.2)	99 (12.9)	93 (12.4)	93 (13.2)	83 (13.2)	83 (15.8)	71 (12.2)	72 (13.9)
Cervical flexion	60 (12.6)[b]	72 (13.5)	68 (12.4)	66 (12.3)	64 (10.5)	59 (10.8)[b]	56 (10.2)	55 (12.0)	53 (10.6)
Cervical extension	59 (19.5)[c]	82 (16.0)	80 (19.2)	67 (13.8)[c]	73 (15.3)	58 (13.1)[c]	61 (14.7)	40 (12.3)[c]	43 (13.3)

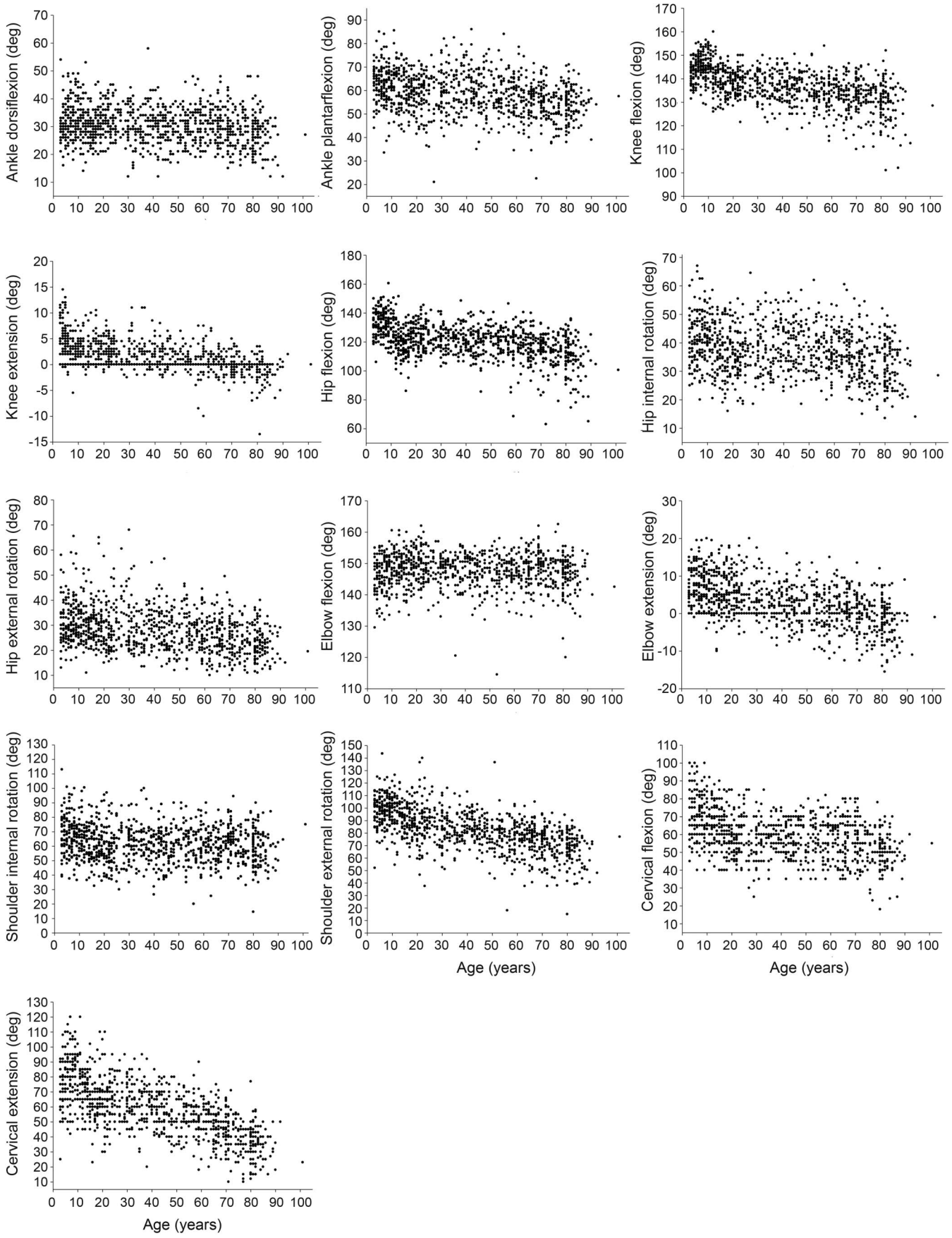
[a] Mean values (SD).
[b] Significant ($p < 0.01$).
[c] Significant sex differences ($p < 0.05$).

adults and in 12 joints of older adults ($p < 0.05$). Greater body mass and waist circumference were associated with a decrease in joint flexibility from 10 years of age. The correlation between height and joint flexibility was strongest in adolescents, where taller individuals demonstrated less joint range of motion. From adolescence to older adulthood, a more pronated foot posture was associated with a greater range of ankle dorsiflexion. Lower limb alignment did not demonstrate any significant correlations, beyond very weak associations, with measures of flexibility in any age category. In children, 2 multiple regression models (see table e-4) reached significance (knee extension and neck extension) and revealed height as the most significant predictor. Age, waist circumference, and height were the strongest independent predictors of flexibility in adolescents. For all adults older than 20 years, age, sex, and waist circumference were the strongest predictors of joint flexibility.

DISCUSSION This study has established a comprehensive reference dataset of isometric muscle strength and joint flexibility in 1,000 healthy people aged 3–101 years. The associations between strength and flexibility measures with demographic and anthropometric variables within different age categories identified some important relationships. As expected, there is a highly significant increase in strength of all muscle groups as children rapidly develop through to early adulthood. From adulthood, this relationship changes and a decrease in muscle strength with aging starts to occur; by older adulthood, all muscle groups demonstrate loss of strength with aging. From 10 years of age, a time of life coinciding with rapid growth and maturation, males are significantly stronger in all measures. In contrast, joint flexibility demonstrates a steady decline with age and no meaningful difference between males and females.

Our results are consistent with previous studies investigating isometric muscle strength in children[8,9] and adults.[12–14] However, direct comparison is limited by differences in age range, sample size, and muscle groups evaluated. Some studies report body mass as the strongest correlate with muscle strength in children,[8–11] while others demonstrate as we did that height showed the strongest relationship and was the most significant predictor of strength.[23,24] In adults, a decline in strength was most strongly associated with aging in 5 muscle groups (hand grip, ankle dorsiflexors, shoulder external rotators, knee flexors, and extensors), while in older adulthood all muscle groups demonstrated a significant decline in strength associated with aging. These results suggest a muscle-specific response to aging during adulthood and that generalized weakness does not occur until older adulthood. This highlights the importance of using age- and sex-matched reference data for specific muscle groups to avoid overrepresentation or underrepresentation of the force capabilities of a particular muscle group. Similar relationships between aging and muscle weakness have been reported in a limited number of adult studies.[12–14]

Figure 2 Scatterplots of joint flexibility vs age for 1,000 children and adults



The association between waist circumference and strength and flexibility has not been reported previously. Waist circumference was identified as a significant predictor of flexibility in adolescents, adults, and older adults and of strength in children. Epidemiologic studies have identified an association between waist circumference and tendon pathology,[25] with preliminary evidence supporting either a mechanical effect (due to increased load) or systemic effect (due to circulating lipids).[26] The influence of adiposity on localized musculo-tendinous tissues in neuromuscular disorders will be an important factor to evaluate with the increasing rates of obesity in society.

Few studies report normative reference values for flexibility in children. Our normative reference values for adults are consistent with the literature.[3,4,7] We identified only one sex difference in the flexibility of children (namely hip internal rotation), and only small differences (2°–6°) between men and women from adolescence through to older adulthood. As such, sex does not seem to have a clinically important effect on the joint flexibility of healthy adolescents and adults. There is no consensus in the literature regarding sex differences and flexibility; some studies report, as we have, that there is no clinically relevant difference,[4] while others report sex differences.[5] We identified a linear decrease in joint flexibility associated with advancing age, consistent with the adult literature.[3] It is likely that in healthy individuals, joint flexibility declines gradually and steadily with age and a substantial or sudden decline should be considered indicative of an underlying pathology.

Studies characterizing the functional decline and rate of progression of neuromuscular disorders such as amyotrophic lateral sclerosis,[27] Duchenne muscular dystrophy,[28] and Charcot-Marie-Tooth disease[29] depend on hand-held dynamometry to capture and track relevant changes in muscle strength. Access to reliable and expansive normative reference values and associated age- and sex-matched *z* scores are necessary to accurately and precisely quantify response to new interventions and to establish minimum clinically important differences.

This study is not without limitation. Participants were recruited through convenience sampling methods and with the exclusion criteria of conditions affecting physical performance may have resulted in a population that were particularly healthy and physically capable for their age. While the mixed ethnicity of our sample is reflective of the Australian population, the ethno-geographic variation in strength and flexibility measures could not be established. The cross-sectional study design was effective in achieving our study aim of generating a reference dataset of strength and flexibility across the lifespan; however, the direction of some of the cause and effect relationships can only be identified in longitudinal studies that track the changes in these measures over time. Ankle plantarflexion strength in healthy adolescents and adults can only ever be estimated with hand-held dynamometry due to the very high force capability (often exceeding 1,000 N).[30] The reported reference values for ankle plantarflexors are likely to underestimate the force capabilities of this muscle group, and values should be used as a lower threshold for weakness in patients with neuromuscular and other neurologic disorders. Finally, the strength and flexibility reference values are specific to the Citec hand-held dynamometer and Baseline goniometer and inclinometer and may not be interchangeable with data obtained from other devices.

The normative reference data generated from this study can be used to determine the presence and extent of impairments associated with neuromuscular disorders and to monitor disease progression over time. The reference values and associated age- and sex-matched *z* scores can be used to develop outcome measures with enhanced precision and responsiveness to be used in clinical trials for neuromuscular and other neurologic disorders.

AUTHOR CONTRIBUTIONS

Marnee J. McKay: study design, data collection, analysis and interpretation, drafting and revising the manuscript. Jennifer N. Baldwin: study design and data collection. Paulo Ferreira: study design and revising the manuscript. Milena Simic: study design and revising the manuscript. Natalie Vanicek: study design and revising the manuscript. Joshua Burns: study conceptualization and design, data interpretation, drafting and revising the manuscript.

ACKNOWLEDGMENT

The authors thank John Eisenhuth for technical assistance, Ray Patton for calibration of the hand-held dynamometer, and the 1,000 volunteers.

STUDY FUNDING

Study funded by National Health and Medical Research Council of Australia Centre for Research Excellence in Neuromuscular Disorders (NHMRC 1031893) and the Australian Podiatry Education and Research Foundation.

DISCLOSURE

The authors report no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

Received July 14, 2016. Accepted in final form September 22, 2016.

REFERENCES

1. Lunn MP, Van den Bergh PY. Outcome measures in neuromuscular disease: is the world still flat? J Peripher Nerv Syst 2015;20:255–259.
2. Mercuri E, Messina S, Pane M, Bertini E. Current methodological issues in the study of children with inherited neuromuscular disorders. Dev Med Child Neurol 2008;50:417–421.
3. Roach KE, Miles TP. Normal hip and knee active range of motion: the relationship to age. Phys Ther 1991;71:656–665.
4. Hallaceli H, Uruc V, Uysal HH, et al. Normal hip, knee and ankle range of motion in the Turkish population. Acta Orthop Traumatol Turc 2014;48:37–42.

5. Sengupta P, De S, Pal A, Maity P, Banerjee M, Dhara PC. Variation of range of joint motion in Bengalee (Indian) healthy adult subjects. J Life Sci 2012;4:123–133.
6. Boone DC, Azen SP. Normal range of motion of joints in male subjects. J Bone Joint Surg Am 1979;61:756–759.
7. Gunal I, Kose N, Erdogan O, Gokturk E, Seber S. Normal range of motion of the joints of the upper extremity in male subjects, with special reference to side. J Bone Joint Surg Am 1996;78:1401–1404.
8. Backman E, Odenrick P, Hendriksson KG, Ledin T. Isometric muscle force and anthropometric values in normal children aged between 3.5 and 15 years. Scand J Rehab Med 1989;21:105–114.
9. Beenakker E, Van der Hoeven J, Fock J, Maurits N. Reference values of maximum isometric muscle force obtained in 270 children aged 4–16 years by hand-held dynamometry. Neuromuscul Disord 2001;11:441–446.
10. Eek MN, Kroksmark A-K, Beckung E. Isometric muscle torque in children 5 to 15 years of age: normative data. Arch Phys Med Rehabil 2006;87:1091–1099.
11. Hébert LJ, Maltais DB, Lepage C, Saulnier J, Crête M. Hand-held dynamometry isometric torque reference values for children and adolescents. Pediatr Phys Ther 2015;27:414.
12. Andrews AW, Thomas MW, Bohannon RW. Normative values for isometric muscle force measurements obtained with hand-held dynamometers. Phys Ther 1996;76:248–259.
13. Bäckman E, Johansson V, Häger B, Sjöblom P, Henriksson K. Isometric muscle strength and muscular endurance in normal persons aged between 17 and 70 years. Scand J Rehab Med 1995;27:109–117.
14. Bohannon RW. Reference values for extremity muscle strength obtained by hand-held dynamometry from adults aged 20 to 79 years. Arch Phys Med Rehabil 1997;78:26–32.
15. Meldrum D, Cahalane E, Conroy R, Fitzgerald D, Hardiman O. Maximum voluntary isometric contraction: reference values and clinical application. Amyotroph Lateral Scler 2007;8:47–55.
16. Van der Ploeg R, Fidler V, Oosterhuis H. Hand-held myometry: reference values. J Neurol Neurosurg Psychiatry 1991;54:244–247.
17. Danneskiold-Samsøe B, Bartels E, Bülow P, et al. Isokinetic and isometric muscle strength in a healthy population with special reference to age and gender. Acta Physiol 2009;197:1–68.
18. Hogrel J-Y, Payan CA, Ollivier G, et al. Development of a French isometric strength normative database for adults using quantitative muscle testing. Arch Phys Med Rehabil 2007;88:1289–1297.
19. The National Isometric Muscle Strength (NIMS) Database Consortium. Muscular weakness assessment: use of normal isometric strength data. Arch Phys Med Rehabil 1996;77:1251–1255.
20. McKay MJ, Baldwin JN, Ferreira P, et al. 1000 Norms Project: protocol of a cross-sectional study cataloguing human variation. Physiotherapy 2016;102:50–56.
21. Redmond AC, Crosbie J, Ouvrier RA. Development and validation of a novel rating system for scoring standing foot posture: the Foot Posture Index. Clin Biomech 2006;21:89–98.
22. Winter DA. Biomechanics and Motor Control of Human Movement. New York: John Wiley & Sons; 2009.
23. Hogrel JY, Decostre V, Alberti C, et al. Stature is an essential predictor of muscle strength in children. BMC Musculoskel Disord 2012;13:176.
24. Macfarlane TS, Larson CA, Stiller C. Lower extremity muscle strength in 6-to 8-year-old children using hand-held dynamometry. Pediatr Phys Ther 2008;20:128–136.
25. Gaida JE, Ashe MC, Bass SL, Cook JL. Is adiposity an under-recognized risk factor for tendinopathy? A systematic review. Arthritis Care Res 2009;61:840–849.
26. Scott A, Zwerver J, Grewal N, et al. Lipids, adiposity and tendinopathy: is there a mechanistic link? Crit Rev Br J Sports Med 2015;49:984–988.
27. Shefner JM, Liu D, Leitner ML, et al. Quantitative strength testing in ALS clinical trials. Neurology 2016:87:617–624.
28. Kirschner J, Schessl J, Schara U, et al. Treatment of Duchenne muscular dystrophy with cyclosporine A: a randomised, double-blind, placebo-controlled multicentre trial. Lancet Neurol 2010;9:1053–1059.
29. Cornett KM, Menezes MP, Bray P, et al. Phenotypic variability of childhood Charcot-Marie-Tooth disease. JAMA Neurol 2016;73:645–651.
30. Kelln BM, McKeon PO, Gontkof LM, Hertel J. Hand-held dynamometry: reliability of lower extremity muscle testing in healthy, physically active, young adults. J Sport Rehabil 2008;17:160.

Exhibit 24

WORLD RUGBY

Transgender Women Guidelines

Can transgender women play rugby?

- Transgender women who transitioned pre-puberty and have not experienced the biological effects of testosterone during puberty and adolescence can play women's rugby (subject to confirmation of medical treatment and the timing thereof)
- Transgender women who transitioned post-puberty and have experienced the biological effects of testosterone during puberty and adolescence cannot currently play women's rugby
- Transgender women can play mixed-gender non-contact rugby
- World rugby are committed to ongoing evaluation of the guidelines and will remain current on all published research that pertains to the biological and physiological implications of testosterone suppression, with a formal review of the Guideline every three years. In support of this, World Rugby will prioritise support for high quality research projects on transgender rugby players, as part of this commitment to evidence-based guidelines.

Why can't transgender women play women's rugby?

Effects of testosterone

re reference is made to "females" and "males" to explain the effects of testosterone, the references are used to diffe

Testosterone is an androgenic-anabolic hormone whose functions include reproductive maturation, along with the genesis of male secondary sex characteristics. From puberty onwards, testosterone levels increase 20-fold in males, but remain low in females, resulting in circulating testosterone concentrations at least 15 times higher in males than in females of any age [1,2]. Among the biological changes initiated by testosterone and its derivatives are:

- Larger and denser lean muscle mass [3,4];
- Greater force-producing capacity of skeletal muscle [5,6];
- Stiffer connective tissue [7];
- Reduced fat mass and different distribution of body fat and lean muscle mass [3];
- Longer, larger and denser skeletal structure [8,9];
- Changes to cardiovascular and respiratory function that include higher haemoglobin concentration, greater cross-sectional area of the trachea and lower oxygen cost of respiration (as described in [1,10-12]).

Collectively, these biological differences account for large sporting performance differences between males and females. These include gaps between 9% and 15% for running, swimming and jumping events [13], between 15% and 35% for functional tasks like kicking, throwing, bowling and weightlifting, and in excess of 50% for tasks that involve upper body force production [10], since the biological effects of testosterone creates disproportionately greater strength on their upper compared to lower body, while females show the inverse [14,15]. In weight-lifting events, for instance, even when matched for mass and stature, males lift approximately 30% more weight than females. Evaluated differently, males are able to lift weights similar to females who weigh 30% to 40% more than them [10]. Functional movements such as explosive jumping are similarly larger in elite males than females, with approximately 30% more power generated during a counter movement jump [10].

The result of these biological differences is that males outperform females in all sporting activities where speed, size, power, strength, cardiorespiratory and anthropometric characteristics are crucial determinants of performance. This is true for many thousands of boys and men who have undergone a testosterone-

performance differences varies depending on the contributions made by each of the biological variables to performance, and indeed, some may be detrimental to performance in some events (mass during endurance running or cycling events, for example). Generally, however, there is no overlap in performance between males compared to females at all matched levels of competition from high school to the elite level The performance disparity is illustrated by the observation that thousands of teenage boys and adult males are able to outperform the very best biological females every year [13].

Similar performance differences between males and females have been described in non athletically trained individuals. Males have muscle mass 30% to 40% greater than females [4], maximal cardiorespiratory capacities (VO2max) 25% to 50% greater than in females [17], cardiovascular parameters between 11% and 43% greater than in females, lower limb strength approximately 50% higher than in females across the lifespan, and upper body strength 50% to 100% higher than in age matched females [6]. Even when elite females, trained in sports where grip strength is an important component of performance Uudo and handball), do not outperform untrained males in a grip strength task, with the very best female performance corresponding to approximately the 58 th percentile for males, and a 26% advantage for untrained males compared to typical elite females Punching performance, a composite movement reliant on strength, power, co-ordination and mass, has been found to be 162% higher in males than in females [18], and 17-year old boys are able to throw a ball further than 99% of adult females [19].

Biological consideration for rugby union

The implications of biological and performance differences for rugby are two-fold. First, significant differences in strength, size, speed and power have potential consequences for the safety of participants in rugby, where much of the sport involves contacts in the form of tackles, rucks and mauls, as well as numerous periods of high force production during static contests for the ball, such as the scrum and ruck. Given the documented risk of injury in rugby from contact events in particular [20-24], the elevated possibility of all injuries, including serious injury, from large disparities in size, speed, power, and force, is of concern Recent modelling of tackles using validated biomechanical models [25,26] suggests that the

greater risks for smaller and slower players, particularly when size and speed exist in combination.

Given that the typical male player mass is 20% to 40% greater than typical women mass, that males have strength 40% to 80% greater (unadjusted for mass), and that men are 10% to 15% faster than women despite being heavier, the risk of injury created by large imbalances in mass and speed may be considered significant. To explore this, we assessed the range of masses of players at the international level and applied the findings to a biomechanical model to explore possible implications for injury risk should cross-over scenarios occur.

With respect to mass, we documented the range of sizes of elite men's and women's players from the 2011 Rugby World Cup up to the 2019 Rugby World Cup, finding:

- Typical (median) men's players are 41.1% heavier than typical women's players (103 kg vs 73 kg)
- Among forwards, the heaviest 1% of women players are smaller than the typical men's forward (109kg for women vs 112kg for men)
- The heaviest 1% of women's backs are smaller than typical men's backs (89kg vs 92kg)
- The lightest 1% of men's forwards are approximately equal in mass to the heaviest 10% of women's forwards, while the lightest 2% of men's backs are approximately equal to the heaviest 10% of women's backs
- Figure 1 below shows the frequency histograms for men's and women's players in forward and back positions

Figure 1: Frequency histograms of mass of forwards (left panel) and backs (right panel) in elite men's and women's rugby players. Dotted lines indicate the 50th percentile, while dashed lines indicate the 98th percentile for each group.

Implications for injury risk - head injury models

The differences observed between men and women with respects to mass may be combined with differences in speed to create a theoretical framework in which the inertial load and forces faced by smaller and slower player is significantly greater when in contact with a larger, faster player. this model is intended for illustrative purposes and demonstrates the impact of only one variable known to differ between biological males and females - namely mass - on head injury risk, in a basic parametric model, absent force application and complex movements, as a preliminary impact analysis. the principles illustrated by the model would apply to other injuries. The addition of speed, and strength or force exerted during contact would further increase the implications of the findings of this illustrative model, summarized below.

The representative figure below illustrates the concept of mass disparity as a risk of injury for ball carriers. It depicts the linear acceleration (A), angular acceleration (B), neck force (C) and neck moment (D) experienced by ball carriers of different masses when tackled by players with different masses. Using the known masses of international rugby player, the position of the average male (M50) and average female (F50) are plotted on each heat map. F90 shows the scenario where a tackler (T) corresponds to the 90th percentile for women's mass (see Figure 1) tackles a typical female mass ball carrier (BC). X indicates the hypothetical cross-over scenario in which a typical male tackler mass is involved in a tackle against a ball carrier with a typical female mass.

Figure 2. Graphical representations of linear acceleration (A), angular acceleration (B), neck force (C) and neck moment (D) for ball carriers of different masses during tackles by tacklers with different masses. Mso and Fso show the modelled situation when typical/median players tackle one another for men and women, respectively. F90 represents a female ball-carrier with typical mass against a tackler in the heaviest 10% of women's body mass. X denotes the cross-over situation that would hypothetically occur for a tackler at the men's median mass tackling a typical female ball carrier

The modelling shows that a tackle involving players with typical or average mass produces slightly greater accelerations and forces in men (Mso) than in women (Fso). This is a function of the higher mass of men's players. Head and neck kinematic and kinetic variables increase significantly when the heaviest 10% of women's body mass is used for the tackler against a typical ball carrier (F90), but this extreme "within female-bodied" scenario produces smaller kinetic and kinematic outcomes than if the hypothetical cross-over scenario were to occur, where an average male-bodied player is the tackler and the average female-bodied player the ball carrier **(X).** The magnitude of the increase in neck forces, moments and accelerations for the ball carrier is between 20% and 30% for typical cross-over scenario compared to the typical female vs female scenario, and is 10% greater for the male-bodied vs female-bodied crossover scenario than a tackle where the heaviest 10% of women are matched against typical women's mass (F90).

compared to the typical tackle scenario in women's rugby. The magnitude of these extreme head accelerations and neck forces are not seen in women and are created by cross-over of male-bodied players to women's rugby. Similar differences are seen when examining the accelerations and forces for the tackler's head and neck.

The magnitude of the known risk factors for head injury are thus predicted by the size of the disparity in mass between players involved in the tackle. The addition of speed as a biomechanical variable further increases these disparities, which is relevant given that male players weighing 103kg (the median for men) would be expected to run between 10% and 15% faster than typical female players (mass 73kg), and thus considerably faster than female players who are heavier than the median (eg females at the 90 th percentile, Fig 1). This would further compound the disparity created.

Next, it is important to also consider that these models do not account for the ability of players to actively exert force at high rates during tackles. This would be a function of power and strength, which are similarly known to be 30% to 80% greater in biological males than females. When these active applications of force during contact are added to the mass and speed characteristics illustrated and described above, the resultant neck and head forces and accelerations will increase even further, such that the illustrative model shown above depicts the smallest possible risk increase for typical players involved in a tackle as a result of mass alone. The addition of speed and force disparities will increase the magnitude of these risk factors beyond the 20% to 30% we illustrate above.

The implication of these increases is complex to quantify but would result in increased injury risk for the player experiencing the elevated risk outcomes (force and acceleration). This is because head injuries occur when forces and accelerations on the head and neck reach a threshold necessary to cause injury, and which is unique to each tackle situation. A tackle situation that typically produces risk factors within 20% of this threshold would, in the circumstance of a typical male-bodied vs typical female-bodied player illustrated above, be sufficiently increased to cause an injury. The higher risk scenario involving heavier ... bodied players would further increase injury likelihood, since all tackle situations that normally ...ce kinetic and kinematic variables within 40% to 50% of an injury threshold would now exceed it, a

causing head injury.

Finally, it must also be considered that the ability to withstand or tolerate forces on the head and neck are required to avoid brain injury. This is the reason neck strength is critical in injury prevention. Since the strength disparities between males and females is so large, including a 50% lower neck isometric strength in females, the reduced ability of female players to tolerate or withstand the forces in tackles is a further risk factor for injury, including head injury as described above, but relevant to all injuries where the rapid application of force or load are responsible for injury.

Implications for injury risk - scrum forces

The implication of greater mass and force-producing ability in males can be seen in forces measured during scrums in both elite and community level rugby. Research on the forces applied during scrums shows that at the elite level, males produce approximately twice the peak force of females in the scrum. Even at the community level, where peak force is 30% lower than in the elite game, males produce approximately 40% greater peak force during scrums than elite females. Given that force producing and receiving ability is likely to be significantly lower in female community players, the implication is that men's community level rugby scrums will be considerably more forceful than women's community level scrums.

The risk of particularly serious and catastrophic injuries during scrums has led to a number of law changes specifically designed to depower the scrum to reduce injury risk. This risk would be amplified by large mismatches in strength between opposing players, since the force applied must be withstood by a direct opponent. This is an illustration of how mismatches in strength and size are directly responsible for forces that result in injury.

It must be noted that the actual testosterone level, measurable in the body, is not a strong predictor of performance within men and within women [27 29] This is because performance is multifactorial, and testosterone's androgenzing effects contribute to, but do not solely influence the biology and resultant performance outcomes within a group who are able to utilize it. The biological basis for male vs female differences is thus the result of testosterone, but it does not necessarily follow that within men and within women, the hormone is a predictor of performance

Further, differences in the sensitivity to testosterone between individuals mean that a given level of testosterone is not a sensitive or specific predictor of performance within each group (males and females) This is in part because most males have elevated levels and some degree of sensitivity, while the level in females is significantly lower and rarely exceeds even the very low end of the male range [1]. Therefore, in two homogenous groups that are matched for either the presence or absence of a given variable (males and females for the presence or absence of testosterone, in this case), the predictive value of that variable within a group is greatly diminished, the same way that V02max is a significant predictor of running or cycling performance across the whole population, but not within a group of elite marathon runners or cyclists, who are already relatively homogenous for that characteristics [30]. Similarly, height is clearly advantageous for professional basketball, but within the National Basketball Association (NBA), where height has already been selected for and participants are in the extreme upper end of the overall population for that characteristic [31], it becomes a poorer predictor of performance.

However, when the same question -does testosterone predict performance across humans of both sexes - is asked of binary categories (males vs females in sport, rather than within males or females), then the predictive power of testosterone is strong, because "high testosterone" during adulthood is a very reliable indicator that the androgenizing effects of testosterone have occurred earlier during life When understood and assessed this way, testosterone is necessary for peak performance (since the top performers within humans are all male), but it is not sufficient to attain it. It is here that the almost perfect sensitivity of biological sex emerges, since in a ranking list of the top thousand performances in port, every year, every single one will be biologically male

In summary, across all performance levels and ages after puberty, testosterone is primarily (though not exclusively) responsible for very large typical differences in the biology of males and females, and consequently, performances between the sexes. These are summarized in Figure 3 below, which combines the biological differences between males and females with their performance implications, and is reproduced from a recent article currently in review [10].

Figure 3: Summary comparison of biological (left table) and performance (right figure) differences between males and females for a range of biological variables and physical activities/events. Reproduced from Hilton & Lundberg [10]

Given that the women's category exists to ensure protection, safety and equality for those who do not benefit from the biological advantage created by these biological performance attributes, the relevant and **crucial question is whether the suppression of testosterone for a period of 12 months, currently required for transgender women participation in women's sport, is sufficient to remove the biological differences summarized above?**

Current policies regulating the inclusion of transgender women in sport are based on the premise that reducing testosterone to levels found in biological females is sufficient to remove many of the biologically based performance advantages described above. However, peer-reviewed evidence suggests that this is not the case, and particularly that the reduction in total mass, muscle mass, and strength variables of transgender women may not be sufficient in order to remove the differences between males and females, and thus assure other participants of safety or fairness in competition

Based on the available evidence provided by studies where testosterone is reduced, the biological variables that confer sporting performance advantages and create risks as described previously appear to be only minimally affected. Indeed, most studies assessing mass, muscle mass and/or strength suggest that the reductions in these variables range between 5% and 10% (as described by Hilton & Lundberg [10]). Given that the typical male vs female advantage ranges from 30% to 100%, these reductions are small and the biological differences relevant to sport are largely retained

For instance, bone mass is typically maintained in transgender women over the course of at least 24 months of testosterone suppression, with some evidence even indicating small but significant increases in bone mineral density at the lumbar spine [32-34]. Height and other skeletal measurements such as bone length and hip width have also not been shown to change with testosterone suppression, and nor is there any plausible biological mechanism by which this might occur, and so sporting advantages due to skeletal differences between males and females appear unlikely to change with testosterone reduction

With respects to strength, **1** year of testosterone suppression and oestrogen supplementation has been found to reduce thigh muscle area by 9% compared to baseline measurement [35] After 3 years, a further reduction of 3% from baseline measurement occurred [36]. The total loss of 12% over three years of treatment meant that transgender women retained significantly higher thigh muscle size ($p<0.05$) than the baseline measurement of thigh muscle area in transgender men (who are born female and experience female puberty), leading to a conclusion that testosterone suppression in transgender women does not ...e muscle size to female levels [36].

find that **1** year of testosterone suppression to female typical reference levels results in a comparatively modest loss of lean body mass (LBM) or muscle size, with consistent changes between 3% and 5% reduction in LBM after 1 year of treatment (as summarized from source research studies by Hilton & Lundberg [10]).

Muscle force-producing capability is reduced after testosterone suppression, though as appears to be the case for muscle/lean mass, these reductions are considerably smaller in magnitude that the initial male-vs-female differences in these variables. For instance, hand-grip strength was reduced by 7% and 9% after **1** and 2 years, respectively, of cross hormone treatment in transgender women [39], and by 4% in 249 transwomen after 1 year of gender-affirming treatment, with no variation between different testosterone levels, age or BMI tertiles [45]. Transgender women retained a 17% grip-strength advantage over transgender men at baseline measurement, with a similarly large, retained advantage when compared to normative data from a reference or comparison group of biological females.

Most recently, Wiik et al found that isokinetic knee extension and flexion strength were not significantly reduced in 11 transgender women after 12 months of testosterone suppression, with a retained advantage of 50% compared to a reference group of biological females and the group of transgender men at baseline [41]. This absence of a reduction in strength occurred in conjunction with a 4% to 5% reduction in thigh volume, and no difference in the contractile density of the muscle, which suggests that the reduction of testosterone for a period of a year had no effect on the force-producing capacity per unit of cross sectional area [41], a variable that is known to be higher in males than females.

In conclusion, longitudinal research studies that have documented changes in lean mass, muscle mass/area and strength show consistently that small decreases occur as a result of testosterone suppression, with a resultant relatively large retained advantage in these variables compared to a group of biological females.

Testosterone exerts significant biological effects on biological males during puberty and adolescence. This creates large differences in strength, mass, speed, power, and endurance capacity. In turn, these create player welfare concerns and performance inequality in rugby, given the importance of physical contact and strength in the sport. Longitudinal research studies on the effect of reducing testosterone to female levels for periods of 12 months or more do not support the contention that variables such as mass, lean mass and strength are altered meaningfully in comparison to the original male-female differences in these variables. The lowering of testosterone removes only a small proportion of the documented biological differences, with large, retained advantages in these physiological attributes, with the safety and performance implications described previously. There is currently no basis with which safety and fairness can be assured to biologically female rugby players should they encounter contact situations with players whose biologically male advantages persist to a large degree.

While there is overlap in variables such as mass, strength, speed and the resultant kinetic and kinematic forces we have modelled to explore the risk factors, the situation where a typical player with male characteristics tackles a typical player with female characteristics increases the magnitude of known risk factors for head injuries by between 20% and 30%. In the event of smaller female players being exposed to that risk, or of larger male players acting as opponents, the risk factors increase significantly, and may reach levels twice as large, at the extremes. The basis for regulation is the typical scenario, though risk mitigation must be mindful of the potential for worst-case scenarios that may arise. Both are deemed unacceptably high, because they would result in a number of tackle situations that currently lie beneath a threshold required to cause injury increasing to exceed that threshold.

Thus, it is on the basis of male vs female biological differences, combined with no evidence for removal of their implications for safety and performance, that the guideline is that trans women should not compete in women's rugby.

It is acknowledged that the published studies currently available on testosterone suppression and physiological changes (compiled and described in Hilton and Lundberg, 2020 and reviewed individually in the proposed policy document) have been conducted in untrained transgender women. This invites questions over the validity and generalizability of the studies to a sports-playing population.

This is a valid question, and it is acknowledged that research is required to fully address questions arising out of this limitation. World Rugby is committed to supporting high quality research proposals in this area, should they be submitted as part of World Rugby's Research programme.

However, this limitation can also be assessed within an understanding of the physiological implications of trained compared to untrained individuals undergoing testosterone suppression. The application of insights from complementary studies leads to a conclusion that the available research is in fact sufficient to arrive at firm conclusions about safety, performance and retained advantages, and thus the recognized limitations are not sufficient to refrain from drawing a conclusion on the likely implications of the transgender research for athletes.

In assessing this issue, two primary questions may be asked:

1. How would training undertaken during the process of testosterone suppression affect the changes observed in muscle and lean body mass, and strength variables, compared to studies done in individuals who do not perform training?
2. How would training prior to a period of testosterone suppression influence:
1. The baseline or pre-suppression measures for muscle mass and strength in transgender women, and thus the differences in these variables compared to a reference or control group of biological women (cisgender women)?
2. The likely "end-point" for muscle and lean body mass as well as strength after the testosterone suppression for a period of at least twelve months, once again compared to a reference or comparison of cisgender women?

Training during the intervention to lower testosterone levels can reduce, eliminate, and even reverse any losses in muscle and lean body mass, muscle volume, and muscle strength. This is supported by evidence from various study models in which biological males reduce testosterone to within the female range, and are able to maintain or even increase these physiological variables through training [46-48].

> The implication is that any performance decline as a result of androgen deprivation is minimized or eliminated, and so the studies cited in support of the World Rugby Guideline, while conducted on non-training individuals, establish the minimum possible retained advantage for trans women. That is, they establish that in the absence of training during testosterone suppression, an advantage is retained compared to cisgender women. That advantage is either the same, or very plausibly increased as a result of training.

Training prior to the intervention will cause increased muscle mass and strength variables at baseline. This means that the initial or "pre-suppression" differences in these variables compared to biological females will be greater than in an untrained trans woman. This rebuts the assertion that trans women are weaker, less muscular and thus more similar to biological females at baseline, within a sporting context, since the transgender woman being considered by World Rugby is much more likely to be trained (or will train once transition begins, as described above).

> Further, once the period of testosterone suppression begins, then the degree to which muscle mass and strength decreases may be either the same or relatively greater in the trained trans women as a result of this higher baseline. Even if the relative loss of muscle mass and strength are higher than in untrained trans women, it is inconceivable, and even physiologically impossible, that a pre-trained athletic trans woman is going to lose so much muscle mass and strength that they end at a point where they are less muscular/lean and weaker than a theoretically untrained (and even 'self-starved') transgender woman.

advantage for a pre-trained trans woman. The effect of training can only be to increase this value or to achieve the same value of X percent retained advantage, but it cannot reduce it further, unless one argues that a trained trans woman will lose so much lean mass and strength that they end up weaker and less muscular than a completely non-athletic individual.

Finally, it is relevant that studies comparing untrained biological males and highly trained females, males retain an advantage despite the training status of biological females. For instance, in a study on grip strength, the strongest elite athletically trained females in sports where grip strength is a performance advantage Uudo and handball) are only as strong as untrained biological males at the 58th percentile, with a 26% difference in strength between typical elite females and typical untrained males [49]. Similarly, Morrow & Hosler (1981) found that untrained college-aged males were more than twice as strong as trained female basketball and volleyball players in a bench press task, with the top 5% strongest trained females equal in strength to the weakest 14% of untrained males This establishes that pre-trained biological females can increase strength beyond that of untrained females, but still do not compare to untrained biological males.

The implication is also that since even typical untrained biological males have a large strength advantage compared to elite and trained females, studies that have documented only small reductions in strength and thus persistence of strength advantages with androgen deprivation in untrained biological males (as in Kvorning et al [46], Chen et al [47] and in studies on transgender women cited herein) should be considered relevant for establishing the smallest possible retained advantage that would exist in the absence of training. As described above, and in studies where training is conducted while testosterone is suppressed [46-48], the advantage will only remain this size or increase.

Finally, it is also recognized that not all sports are affected similarly by the variables we have weighted as crucial for rugby (size, strength, speed, power). Indeed, in some sports, excess mass may be disadvantageous, and thus the model for retained advantage and persistent risk may present differently for different physical activities.

physiological and performance advantages and those to whom it does not are removed sufficiently to enable participation of transgender women in women's rugby. At the present time, however, based on the best published scientific evidence, that position is unsupported.

The referenced research used to support this position can be viewed **here**.

Conclusion - Testosterone, Welfare and Performance

Having considered all of the currently available information, the working group determined that the best evidence **currently** available means that those who experienced the biological effects of testosterone during puberty and adolescence cannot safely or fairly compete in women's rugby. That means that currently, transgender women may not compete in women's rugby.

World Rugby is committed to encouraging transgender people to remain involved with rugby and is currently funding research to continue to review any evidence that may emerge to enable the participation of transgender women in women's rugby. Details of the research currently underway, along with details of how to apply for research funding for those who may be interested, is available **here**.

How do I stay involved in rugby if I can no longer play in the category that I want to?

World Rugby acknowledges that the introduction of this Guideline will mean that some players can no longer play in the category that they want to. It is possible that will change in the future and World Rugby is funding research to try to find out if there are ways to allow that safely and fairly (see **here** for details). In the meantime, there are many other ways to stay involved with rugby:

- Other forms of the game: Many forms of non-contact Rugby exist such as: Tag; Touch; Flag etc all have

courses for coaches of children, adolescents, and adults. All courses are open to any participant.

- Refereeing: For many people who may not be able to play, refereeing is a viable alternative to stay close to the game. World Rugby and its member Unions offer several introductory courses and a pathway exists in all Unions for fast-tracking talented
- Administration: All clubs rely on volunteer administrators. As individuals enter the latter stages of the long-term participant model, then administration becomes a realistic outlet for many. A number of Unions have dedicated support resources for individuals who wish to pursue this path of staying involved.

World Rugby is currently exploring the possibility of an "open category" of rugby in which any player could play, regardless of gender identity. World Rugby has committed to exploring this option with its Unions, Associations, International Rugby Players, and trans advocate groups including Gendered Intelligence and International Gay Rugby.

What if I have concerns about safety or fairness relating to someone I am playing with or against?

It is important to note that many people do not meet cultural or local norms or stereotypes related to the expression of gender identity. All players and Unions ought to take care to consider this when raising any concerns about another player. In the event that a player or Union has a genuine concern about safety or fairness in relation to another player, the concern should be dealt with as follows:

1. The concerned person should raise their concerns with their Union's Chief Medical Officer (CMO).
2. The Union's CMO should carefully consider the concerns raised, in the context of all of the known facts and if having done so, the CMO determines that the concerns are not frivolous or vexatious, the CMO should contact the World Rugby CMO setting out the basis for the
3. The World Rugby CMO will engage with the CMO of the Union of the player about whom the concerns h been raised, ensuring confidentiality for the player and involved team-mates and opponents ghout the

5. In some circumstances, such appropriate actions may include a recommendation that a standardised endocrinological assessment be performed [Appendix].

6. For the avoidance of doubt, no player should or would be forced to undergo any medical or other assessment. It is a player's responsibility to decide on whether he or she wishes to proceed with any assessment. However, it should be noted that deciding not to participate in an assessment, having been requested to do so, may have consequences in terms of the player's eligibility to participate in the category of competition that is consistent with his/her/their gender identity, since it may not be possible to determine whether issues of safety or fairness arise without such assessment.

Exhibit 25

STATE OF ARIZONA
OFFICE OF THE GOVERNOR

DOUGLAS A. DUCEY
GOVERNOR

EXECUTIVE OFFICE

March 30, 2022

The Honorable Katie Hobbs
Secretary of State
1700 W. Washington, 7th Floor
Phoenix, AZ 85007

RE: Senate Bill 1138 irreversible gender reassignment surgery; minors & Senate Bill 1165 interscholastic; intramural athletics; biological sex

Dear Secretary Hobbs,

Today I signed S.B. 1138 and S.B. 1165, legislation to protect participation and fairness for female athletes, and to ensure that individuals undergoing irreversible gender reassignment surgery are of adult age. This legislation is common-sense and narrowly-targeted to address these two specific issues — while ensuring that transgender individuals continue to receive the same dignity, respect and kindness as every individual in our society.

S.B. 1138 delays any irreversible gender reassignment surgery until the age of 18. The reason is simple, and common sense – this is a decision that will dramatically affect the rest of an individual's life, including the ability of that individual to become a biological parent later in life.

Distinguishing between an adult and a child in law, as this bill does, is not unique. Throughout law, children are protected from making irreversible decisions, including buying certain products or participating in activities that can have lifelong health implications. These decisions should be made when an individual reaches adulthood. Further, many doctors who perform these procedures on adults agree it is not within the standards of care to perform these procedures on children.

The irreversible nature of these procedures underscores why such a decision should be made as an adult, not as a child, and further supports the importance of this legislation.

S.B. 1165 creates a statewide policy to ensure that biologically female athletes at Arizona public schools, colleges, and universities have a level playing field to compete. This bill does not deny student-athletes the eligibility to play on teams not designated as "female," and it doesn't impact club sports leagues offered outside of schools. Every young Arizona athlete should have the

opportunity to participate in extracurricular activities that give them a sense of belonging and allow them to grow and thrive.

This legislation simply ensures that the girls and young women who have dedicated themselves to their sport do not miss out on hard-earned opportunities including their titles, standings and scholarships due to unfair competition. This bill strikes the right balance of respecting all students while still acknowledging that there are inherent biological distinctions that merit separate categories to ensure fairness for all.

Sincerely,

Douglas A. Ducey
Governor
State of Arizona

cc: The Honorable Karen Fann
The Honorable Rusty Bowers
The Honorable Warren Petersen
The Honorable Nancy Barto

EXHIBIT 4

WILENCHIK & BARTNESS
A PROFESSIONAL CORPORATION

Jane Doe, by her next friends and parents, Helen Doe and James Doe;
and Megan Roe, by her next friends and parents, Kate Roe and Robert Roe

.v.

Thomas C. Horne, in his official capacity as State Superintendent of Public Instruction;
Laura Toenjes, in her official capacity as Superintendent of Kyrene School District;
Kyrene School District;
The Gregory School;
Arizona Interscholastic Association, Inc.

Case 4:23-cv-00185-JGZ

Expert witness statement
Emma Hilton, PhD

1. Qualifications and experience

1.1. I am Emma Hilton. I am a postdoctoral researcher in developmental biology—the study of how embryos grow and how individuals mature—at the University of Manchester, UK, a world top 50 university.[1] My short-form academic curriculum vitae is attached in **Appendix 1**.

1.2. In 1999, I received my Bachelor of Science degree from the University of Warwick, UK, where I studied Biochemistry. My final year dissertation described research to identify a genetic cause of Sotos syndrome, a genetic disorder characterised by, among other features, prenatal and childhood bone overgrowth, leading to unusually-early peak height velocity, increased stature during childhood, and concurrent advanced bone age.[2] In 2004, I received my Doctor of Philosophy degree from the University of Warwick, UK, having identified a gene regulatory mechanism that integrates molecular growth signals to specify the future tissue development of a particular region of the very early "ball-of-cells" stage vertebrate embryo.[3,4]

1.3. Since 2004, I have been employed as a developmental biologist at the University of Manchester, UK. My developmental biology career has focussed on the molecular mechanisms underpinning inherited genetic disorders in humans, including—but not limited to—those that differently affect males and females and those that affect neuromuscular development during embryo development.[5] I am currently employed in a research programme to uncover the molecular development of the skin surface in tadpoles, which is the animal model I have systematically exploited to understand human development and disease.

1.4. I have authored over 20 peer-reviewed publications in developmental biology and genetics journals, and have received over 1300 citations. My h-index is 17.[6] I have contributed a chapter entry to a key medical textbook on genetic disorders.[7] In 2007, I received the honour of being named as an Outstanding Young Investigator by the European Society of Human Genetics for my research on a sex-linked genetic disorder that causes first-trimester death in male fetuses.[8]

1.5. Although not employed in a teaching role, I deliver an annual lecture to undergraduate medical students in genetic disorders, inheritance and the ethics of medical screening. I have previously delivered teaching to ophthalmology Masters students in eye development and genetic disorders of the eye, and to undergraduate dentistry students on craniofacial disorders.

1.6. Developmental biology is not simply the study of specific processes in specific species (for example, as part of my current collaborative research, how a nerve makes a junction with a developing block of muscle to generate a functional movement unit.) The discipline of developmental biology operates on common principles: how regions are zoned; how cells "talk" to each other; how tissues and organs interact in synergistic or exclusive patterns; how such interactions proceed. These common principles apply to

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[1] https://www.manchester.ac.uk/study/experience/reputation/rankings/
[2] https://www.genomicseducation.hee.nhs.uk/genotes/knowledge-hub/sotos-syndrome/
[3] Rex et al., 2002. Multiple interactions between maternally-activated signalling pathways control Xenopus nodal-related genes. Int J Dev Biol 46: 217-226.
[4] Hilton et al., 2003. VegT activation of the early zygotic gene Xnr5 requires lifting of Tcf-mediated repression in the Xenopus blastula. Mech Dev 120(10): 1127-1138.
[5] https://www.research.manchester.ac.uk/portal/emma.hilton.html
[6] https://scholar.google.com/citations?user=A8zl2ggAAAAJ&hl=en
[7] Hilton et al., 2016. "The BCL6 corepressor (BCOR) and oculofaciocardiodental syndrome." In Epstein's Inborn Errors of Development: The Molecular Basis of Clinical Disorders of Morphogenesis. Oxford University Press, Oxford, UK.
[8] https://www.eshg.org/index.php?id=102

multiple events in the global development of all species. A solid understanding of such principles—as I have acquired over my 20-year career—permits any developmental biologist to quickly build a picture of developmental events outside of their specific research programme. The differentiation, development and patterning of the reproductive system and the physical changes induced during maturation are no exception for a trained developmental biologist.

1.7. Over the past six years, I have deepened my academic knowledge of physical sex development in many species, particularly humans. Notably, my active research has always involved extensive sexing and breeding of animals, dissecting reproductive organs like male testes (frogs) and the female uterus (mice), and understanding reproductive issues in my animal colonies (for example, the loss of male sex characteristics with aging in frogs). As part of my previous research in a sex-linked genetic disorder, I have routinely visualised and analysed sex chromosome conformation in mice and humans.[9]

1.8. My expertise in human sex development is increasingly recognised in an academic context. In 2021, I was invited by the editor to publish a letter in the official organ of the Royal Academy of Medicine in Ireland, where I argued that, "*Human sex is an observable, immutable, and important biological classification; it is a fundamental characteristic of our species, foundational to many biology disciplines, and a major differentiator in medical/health outcomes.*"[10] I am the invited lead author of a chapter on human sex development in an academic "primer" textbook to be published in August 2023.[11] Titled "Two sexes", this peer-reviewed chapter describes the evolution trajectory of the two sexes in almost all complex species, the development of sexed anatomy in humans, and common myths regarding the phenomenon of sex. Although not yet published, the chapter text is attached in **Appendix 2**. Since 2022, I have delivered a seminar to undergraduate life sciences students in sex development and the long-term effects of sex hormones on the development of the human body.

1.9. During my school years, I competed in interscholastic and regional competitions in judo, track running, netball, field hockey, cross-country and tennis. As an adult, I have completed two marathons. I currently participate in recreational sports, playing netball in single-sex and mixed-sex leagues, and weightlifting with a personal trainer. I am a sports fan.

1.10. The relevance of developmental biology in sports performance has been typically underestimated, particularly in the context of transgender athletes. A long-standing assumption has been that hormonal intervention is sufficient to secure fairness when transgender women were included in female sports. I and Doctor Tommy Lundberg (Karolinska Institutet, SWE) challenged, for the first time in the academic literature, that assumption. In Hilton and Lundberg (2021),[12] the peer-reviewed academic publication most relevant to this expert statement, we, "*review[ed] how differences in biological characteristics between biological males and females affect sporting performance and assess[ed] whether evidence exists to support the assumption that testosterone suppression in transgender women removes the male performance advantage and thus delivers fair and safe competition.*" We concluded that, *"[T]he muscular advantage*

[9] For example, Hilton et al. 2009. BCOR analysis in patients with OFCD and Lenz microphthalmia syndromes, mental retardation with ocular anomalies, and cardiac laterality defects. Eur J Hum Genet 17: 1325–1335.
[10] Hilton et al., 2021. The reality of sex. Ir J Med Sci 190: 1647.
[11] Hilton and Wright, 2023. "Two sexes." In Sex and Gender: A Contemporary Reader. Routledge, Oxford, UK.
[12] Hilton and Lundberg, 2021. Transgender Women in the Female Category of Sport: Perspectives on Testosterone Suppression and Performance Advantage. Sports Medicine 51: 199–214.

enjoyed by transgender women is only minimally reduced when testosterone is suppressed."

1.11. In terms of impact (26th June 2023), we published our review in Sports Medicine, an international leader in sports and exercise medicine research, with a five-year impact factor of 13.671.[13] Our Altmetric score is 5471, and our review is ranked 662 out of 23.9 million academic articles published across all fields.[14] It has already been cited 65 times in the academic literature,[14] and also in scientific media including Nature.[15] Hilton and Lundberg (2021) has been cited in the transgender athlete policies of British Triathlon,[16] British Cycling[17] and World Rugby[18] (which was used to formulate the transgender policies of England Rugby, Scottish Rugby and Welsh Rugby), and cited in the scientific reviews underpinning the policies of Union Cycliste Internationale[19] and World Athletics.[20] It was also cited by the UK Sports Council Equality Group in their influential policy document that highlighted the clash between fairness for female athletes and inclusion of transgender women athletes.[21] In 2022, Hilton and Lundberg (2021) was cited in the US Court of Appeals for the 11th Circuit, by Justice Lagoa in her specially concurring opinion in Adams .v. School Board of St. Johns County, Florida.[22] Also in 2022, we were cited in a literature review on transgender athletes, published by the UK Parliamentary Office of Science and Technology, intended to brief UK Members of Parliament on topical issues.[23] Finally, Hilton and Lundberg (2021) is cited in the findings of the Fifty-fifth Legislature of the State of Arizona in Senate Bill 1165 (SB1165; the legislation relevant to this case).

1.12. In 2021, I was invited to author a policy review by the Canadian Macdonald-Laurier Institute.[24] This policy document is a review of the individual authors' peer-reviewed publications and expert knowledge; it was not itself peer-reviewed by the academic community. In this policy document, we review the importance of sex categories in sport, synthesising knowledge across developmental biology, the physiology of transgender women, and sports philosophy. We conclude that a female category that excludes all males, regardless of gender identity, is philosophically coherent in terms of category definition and necessary to ensure everyone can compete fairly and fully. We argue it is reasonable for female athletes to expect that their rights will be upheld by the institutions and procedures of their sports.

1.13. I have been asked to consult with various UK and international sporting bodies seeking advice on policy formation. Many such meetings have been held under conditions of anonymity. In February 2020, I was invited, alongside world experts in transgender endocrinology, sports science and ethics, by World Rugby to give evidence to the

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[13] https://www.springer.com/journal/40279
[14] https://link.altmetric.com/details/95647691
[15] Photopoulos, 2021. The future of sex in elite sport. Nature 592: S12-15.
[16] https://www.britishtriathlon.org/britain/documents/about/edi/transgender-policy-effective-from-01-jan-2023.pdf
[17] https://www.britishcycling.org.uk/zuvvi/media/Transgender_and_Non-Binary_Policy_-_FAQs.pdf
[18] https://www.world.rugby/the-game/player-welfare/guidelines/transgender/faqs
[19] https://assets.ctfassets.net/761l7gh5x5an/4gHOE5EpVItQux9kf39XYC/5c52616af086bdf2c9731679f213c1cd/The_current_knowledge_on_the_effects_of_gender-affirming_treatment_on_the_markers_of_performance_in_transgender_female_cycli.pdf
[20] Not publicly available.
[21] https://www.uksport.gov.uk/news/2021/09/30/transgender-inclusion-in-domestic-sport; Sports Council Equality Group Guidance for Transgender Inclusion in Domestic Sport, 2021.
[22] https://aboutblaw.com/6fe
[23] https://researchbriefings.files.parliament.uk/documents/POST-PN-0683/POST-PN-0683.pdf
[24] Pike, Hilton and Howe, 2021. Fair Game: Biology, Fairness and Transgender Athletes in Women's Sport. Macdonald-Laurier Institute, Canada.

Transgender Working Group, which was tasked with reviewing their regulations for inclusion of transgender women in female categories in elite international competition.[25] After an extensive, 'mock courtroom/adversarial' consultation process, World Rugby determined that female categories can only be safe and fair if males, regardless of gender identity, are excluded from female categories. During 2021, I was consulted as part of a policy project by the UK Sports Council Equality Group.[26] In July 2022, I was invited to present to the Equality, Diversity and Inclusion Commission of World Triathlon, who subsequently tightened restrictions on transgender women athletes in the female category.[27]

1.14. In December 2021, I participated in an online academic seminar hosted by Sports Resolutions, alongside David Grevemberg, the managing director of the Commonwealth Games Federation.[28] In April 2022, I was invited to speak at the Canadian Academy of Sport and Exercise Medicine 2022 Annual Conference, on the topic of transgender athletes, fairness and eligibility.[29] In November 2022, I was invited to speak at the Royal Academy of Medicine (UK), alongside Richard Budgett, the medical director of the International Olympic Committee.[30] In March 2023, I was invited to speak at the 19th World Congress of the International Academy of Human Reproduction, on the topic of transgender athletes in sports.[31]

1.15. Beyond academic activities, I am a vocal advocate for fairness in female sport, and have presented my research findings and arguments in various formats. In January 2021, I was appointed as a board member of Sex Matters, a UK-based human rights group who lobby for clarity on the protected characteristic of sex in law and in institutions.[32] Examples of my outputs for Sex Matters include formal responses to sports policy consultations.[33] I offer advice and input to other resources produced by employees. I vote on board-level decisions regarding strategy, expenditure, employment decisions and other typical administrative duties. My position with Sex Matters is unpaid and my work is voluntary. I receive compensation for travel, food and accommodation at meetings and events.

1.16. Other examples of advocacy include the first presentation of my research findings and arguments in July 2019 at an event organised by two feminist groups, A Woman's Place UK and FairPlay For Women.[34] In this presentation, I mapped the timeline of policy development by the International Olympic Committee (IOC) with the concurrent scientific data. I was—and remain—strongly critical of the IOC policy development trajectory. In April 2022, I was invited to speak at a private meeting at the UK House of Lords (for which I was compensated for travel costs), and wrote a house-wide briefing pack. I have been invited to consult with athlete groups like the US-based Women's Sports Policy

[25] https://www.world.rugby/news/563437/landmark-world-rugby-transgender-workshop-important-step-towards-appropriate-rugby-specific-policy; World Rugby Transgender Guidelines, 2020.
[26] https://www.uksport.gov.uk/news/2021/09/30/transgender-inclusion-in-domestic-sport; Sports Council Equality Group Guidance for Transgender Inclusion in Domestic Sport, 2021.
[27] https://www.triathlon.org/news/article/transgender_policy_process
[28] https://www.youtube.com/watch?v=TbE9aEo8ypA
[29] https://casem-acmse.org/wp-content/uploads/2020/02/ENG_CASEM-AQMSE-Quebec-2022-CASEM-AQMSE-1.pdf
[30] https://www.mededucare.com/_files/ugd/70d91e_b49fb63fc9574bac9ce9c34bfac298a9.pdf
[31] https://hr2023.humanrepacademy.org/scientific-program/
[32] https://sex-matters.org/about/emma-hilton-phd/
[33] For example: https://sex-matters.org/wp-content/uploads/2021/05/Sex-Matters-British-Cycling-policy-response.pdf
[34] https://www.youtube.com/watch?v=pzg9QtQeIR8

Working Group[35] and the Independent Council on Women's Sport (ICONS).[36] For the latter, I presented at their inaugural event in Las Vegas in June 2022, and I am due to present again in Denver in July 2023. I received compensation for travel, food and accommodation at the inaugural ICONS event.

1.17. I have been interviewed in the UK media on several occasions, including on BBC Radio 4 and BBC Radio 5 Live Sport. I have published opinion pieces in the mainstream media, including the Wall Street Journal (on the harms arising from denial of the biological reality of sex).[37] Most recently, I wrote with Professor David Handelsman, an international expert in the pharmacology of androgens and expert witness for World Athletics.[38]

1.18. I have been asked by the legal team for the Arizona Superintendent of Public Instruction to provide my expert scientific opinion on the need for a protected female sports category, and the loss of fairness for female athletes arising from the inclusion of transgender girls and transgender women in competitive school sports. In preparation for this case, I have read Senate Bill 1165 (SB1165). My understanding of SB1165 is that sports teams within public schools (or in schools engaged in competitive sports against public schools) will be designated by sex as male or female, or designated as mixed-sex. Female-designated teams will exclude male athletes. An effect of SB1165 is the exclusion of transgender girls from teams designated as female-only. I understand that transgender girls are free to participate in male-designated and mixed-sex teams.

1.19. I am currently retained to provide expert scientific opinion for the State of Indiana and the State of Utah. There is no conflict of interest to declare.

1.20. The opinions put forward in this statement are my own, grounded in my education and scientific expertise, and do not necessarily reflect those of my employer, the University of Manchester, UK. I will make no personal, social, sporting or academic gains from the opinion I present here.

1.21. I am being compensated for my time researching and preparing this report at a rate of $400 USD per hour. I will be compensated for deposition at a rate of $450 USD per hour. My compensation does not depend on the outcome of this litigation.

[35] https://womenssportspolicy.org/
[36] https://www.iconswomen.com
[37] https://www.wsj.com/articles/the-dangerous-denial-of-sex-11581638089
[38] https://amp.theaustralian.com.au/sport/what-science-tells-us-about-transgender-women-athletes/news-story/cb8b7a30f68745a3fa65442b7ff15694

2. Summary of expert witness statement

2.1. Male development, driven by both genetics and hormones, delivers structural differences (compared with females) from as early as first trimester gestation. Physical differences between males and females that matter for athletic sports are detectable in utero, during childhood, and then cemented during puberty.

2.2. Male athletic advantage over female peers in adolescence and adulthood is undisputed. In childhood, male athletic advantage over female peers is evident across track and field events from 8 years old onwards Males systematically outperform their female peers from 8 years old at a frequency that is vanishingly unlikely to result by chance.

2.3. Protected female sports categories are justified to protect fairness (and, discipline-dependent, safety) for female athletes, who, by virtue of typical female development, do not benefit from male development and thus male athletic advantage. This includes protected categories for young female athletes.

2.4. The suppression of testosterone post-puberty in transgender women does not appear to affect skeletal proportions and reduces acquired muscle mass by only a modest amount. The sparse evidence regarding musculoskeletal metrics in transgender girls who have blocked or partially-blocked puberty reveals metrics like height far exceeding those of typical females.

2.5. It is my professional opinion that the State of Arizona is justified in protecting fairness for female athletes in interscholastic sports competition by restricting from those female categories transgender girls and transgender women, because those individuals will have acquired male athletic advantage by virtue of biological development, and acquisition of male athletic advantage is not entirely removed by either puberty blockers and/or testosterone suppression post-puberty.

3. Sex and gender identity

3.1. Sex is an evolved system function common to almost all complex life on earth. Across the natural world, the words “male” and “female” pertain to the two specific reproductive functions within a system of sexual reproduction that proceeds via two differently-specialised gamete types. They are words used to describe cells, tissues, organs and/or entire individuals that have a physical role in the contribution of small gametes (like sperm) or large gametes (like ova), respectively, to the next generation. “Male” and “female” describe the biology of reproduction and I use these words as neutral descriptions of reproductive biology.

3.2. In humans (and indeed, in almost all animals and many plants), the two reproductive functions are divided between two classes of individual, with each class possessing a distinct and specialised molecular and anatomical pattern corresponding to one of the two reproductive functions. In humans, there are two sexes.

3.3. During embryonic development in utero, males and females develop sex-specific primary sex characteristics that have evolved to facilitate function during future reproduction. In humans, healthy male anatomy comprises gonads in the form of external testes (also called testicles) that will make sperm, internal genital structures like the vas deferens (that carries sperm from the testicles to penis) and external genitalia in the form of a penis and scrotum. In contrast, healthy female anatomy comprises gonads in the form of internal ovaries that will make eggs, internal genital structures like a uterus and vagina, and external genitalia in the form of a vulva, incorporating the clitoris.

3.4. The various parts of the reproductive anatomy of a healthy baby (gonad type, internal genitalia, external genitalia) develop as a system in a regulated and coordinated sequence of events. The sex of a baby is routinely and reliably learned or observed—not “assigned”, which implies an element of choice or arbitrariness—at birth by visual and palpable[39] assessment of external genitalia, which is a highly-sensitive marker for the whole system.

3.5. The above descriptions of primary sex are standard, appearing in dictionaries,[40] key biology textbooks,[41] academic publications[42] and medical consensus statements like that issued by the Endocrine Society in 2021.[43] By these standard descriptions of sex, transgender girls and transgender women are biologically male and not biologically female.

3.6. Transgender girls and transgender women feel deep distress and discomfort with their male sex (“gender dysphoria”) and claim a sense of identification with the female sex (via “gender identity”). The assertion that “*everyone has a gender identity*” (Shumer declaration, 18) is contradicted by the personal testimonies of people, including myself, who do not experience a gender identity and the delineation of the concept of ‘agender’, which describes “*identifying as having no gender*” (quoted from Shumer declaration in Flack et al. .v. Wisconsin Department Of Health Services).[44] It appears incoherent to

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[39] “Palpable” means, roughly, “detect by touching”. This assessment is typically used to confirm the healthy descent of testes in male babies.

[40] Examples include: Oxford English Dictionary; Merriam-Webster Dictionary.

[41] Examples include: Baresi and Gilbert, 2020. Developmental Biology. Oxford University Press, UK; Wolpert, Tickle and Martinez Arias. Principles of Development. Oxford University Press, UK.

[42] Academic publications defining sex, actively researching sex or incidentally dependent on these understandings of sex are too numerous to consider. For example, a search on the scientific publication database PubMed for only “male [AND] sperm” (that is, not an exhaustive search) retrieves over 100,000 results, including multiple results from Nobel Laureates in Physiology or Medicine, and from a huge array of biology and medical disciplines.

[43] Barghava et al., 2021. Considering Sex as a Biological Variable in Basic and Clinical Studies: An Endocrine Society Scientific Statement. Endocrine Reviews, 42(3): 219-258.

[44] http://files.eqcf.org/wp-content/uploads/2019/04/170-Shumer-Expert-Witness-Report.pdf

argue that everyone has a gender identity while recognising the existence of being 'agender'.

3.7. I am scientifically-neutral to the possibility that "*gender identity has a strong biological basis*" (Shumer declaration, 19 and 22). I do not consider gender identity to be a component of sex, which denotes one's physical reproductive development and reproductive role. Even if it is true that gender identity is in some way biological in basis, gender identity is irrelevant to eligibility for sporting categories based on sex. The premise that, in transgender people, sex "*designation turns out to be inaccurate because it does not reflect the person's gender identity*" (Shumer declaration, 27) creates a contradiction where gender identity is asserted as a feature of sex (Shumer declaration, 26) yet is an identity that exists by reference to one's sex (Shumer declaration, 25, decouples gender identity from "*birth sex*").

3.8. Disorders of sex development (DSDs), where the development of reproductive anatomy is atypical or disrupted,[45] are very rare[46] but frequently used to argue that sex in humans cannot be described as simply male and female. While it is true that, rarely even within DSDs, the sex of some individuals is difficult to classify, this is irrelevant when considering the sex of transgender people, who do not typically have DSDs.

[45] For example: Arboleda et al., 2014. DSDs: genetics, underlying pathologies and psychosexual differentiation. Nature Reviews Endocrinology 10(10): 603-615.

[46] Sax, 2002. How common is Intersex? A response to Anne Fausto-Sterling. Journal of Sex Research 39 (3): 174-178.

4. Sex and somatic growth

4.1. Beyond differences in reproductive anatomy, males and females differ in somatic (non-reproductive) physical characteristics. Somatic differences first emerge in utero, are evident at birth, and are further cemented during puberty.

4.2. Small differences in average body length (measured as head-bottom length) can be detected by ultrasound from the first trimester of pregnancy, when males are already slightly longer than females.[47] Larger average skull diameter in male fetuses at twenty weeks has been reported.[48] Gestational growth charts track not just higher male values for skull diameter but also higher abdominal circumference and estimated fetal weight.[49] Analysis of growth charts[50] for male and female infants reveals that, at birth, males are, on average, slightly longer and heavier than females.

4.3. In a large study of male and female fetuses and newborns, Broer-Brown et al (2016) concluded that, "Sex affects both fetal as well as infant growth. Besides body size, also body proportions differ between males and females with different growth patterns."[51] Although the magnitude of size differences in utero and at birth are small, they are consistently-different between males and females; indeed, sex is considered necessary to clinically assess fetal growth with accuracy.[52]

4.4. Males are consistently 1-2 cm taller than females between 0-10 years old. Boys at 10 years old also have a larger vertebral cross-sectional area (larger spinal columns) than girls.[53] Girls enter puberty earlier than boys, typically around 10 years old, and the growth spurt associated with earlier pubertal onset accounts for taller female height between 10-14 years old. Boys catch up and overtake girls in height at around 14 years old.

4.5. At puberty, both sexes undergo rapid somatic changes as they mature in preparation for reproduction, leading to measurably different adult body shapes ('sexual dimorphism').[54] Many male secondary sex characteristics are rooted in our evolutionary history of male fighting ability, displays of strength and competition for mates[55] and become increasingly evident as puberty progresses.

4.6. When—briefly—considering sexually-dimorphic physical characteristics in males compared with females, adolescent and adult males are typically taller with wider shoulders, longer limbs and longer digits. They have larger and denser muscle mass, reduced fat mass, different distributions of muscle and fat, and stiffer connective tissue.

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[47] Pedersen, 1980. Ultrasound evidence of sexual difference in fetal size in first trimester. British Medical Journal 281(6250): 1253.

[48] Persson et al., 1978. Impact of fetal and maternal factors on the normal growth of the biparietal diameter. Scandinavian Association of Obstetricians and Gynaecologists 78: 21-27.

[49] Schwartzler et al., 2004. Sex-specific antenatal reference growth charts for uncomplicated singleton pregnancies at 15–40 weeks of gestation. Ultrasound in Obstetrics and Gynaecology 23(1): 23-29.

[50] For example: World Health Organisation https://www.who.int/tools/child-growth-standards/standards; Centre for Disease Control https://www.cdc.gov/growthcharts/clinical_charts.htm; Royal College of Paediatrics and Child Health https://www.rcpch.ac.uk/resources/growth-charts

[51] Broere-Brown et al, 2016. Sex-specific differences in fetal and infant growth patterns: a prospective population-based cohort study. Biology of Sex Differences 7: 65.

[52] Galjaard et al., 2019. Sex differences in fetal growth and immediate birth outcomes in a low-risk Caucasian population. Biology of Sex Differences 10: 48.

[53] Gilsanz et al., 1997. Differential Effect of Gender on the Sizes of the Bones in the Axial and Appendicular Skeletons. Journal of Clinical Endocrinology and Metabolism 82(5): 1603-1607.

[54] For example: Darwin, C. *The Descent of Man, and Selection in Relation to Sex*. London: Murray, 1871; Well, 2007. Sexual dimorphism of body composition. Best Practice and Research Clinical Endocrinology and Metabolism 21(3): 415-430.

[55] For example: Morris et al., 2020. Sexual dimorphism in human arm power and force: implications for sexual selection on fighting ability. Journal Of Experimental Biology 223(2): 212365; Puts, 2010. Beauty and the beast: mechanisms of sexual selection in humans. Evolution And Human Behaviour 31(3): 157-175.

They have higher amounts of haemoglobin (the molecule that carries oxygen in blood), and larger hearts and lungs.[56]

4.7. The above is a non-exhaustive list of sexually-dimorphic differences between males and females, which could number into the thousands, and include, for example, the fine architecture of muscle tissue like proportions of cell type (fibre type, stem cell populations), cell morphology (numbers of nuclei, amounts of myoglobin) and some 3000 muscle-specific gene expression differences,[57] to the minutiae of different visual perception, hand-eye coordination and tracking capacity.[58]

[56] Reviewed in: Hilton and Lundberg, 2021. Transgender Women in the Female Category of Sport: Perspectives on Testosterone Suppression and Performance Advantage. Sports Medicine 51, 199–214 (and references therein).

[57] Haizlip et al., 2014. Sex-Based Differences in Skeletal Muscle Kinetics and Fiber-Type Composition. Physiology (30)1: 30-39.

[58] For example: Mathew et al., 2020. Sex differences in visuomotor tracking. Scientific Reports 10: 11863.

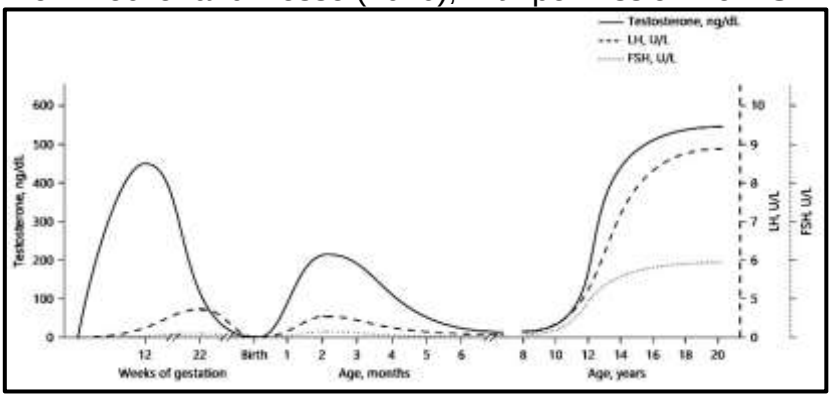
5. Genetics, hormones and development

5.1. Sex differentiation is initiated in utero by the presence or absence of a gene called SRY, typically carried on the Y chromosome, and triggering bipotential gonad development into testes or ovaries in males or females, respectively.[59] The developing gonads, in conjunction with other tissues, establish sex-specific hormonal milieu that, in concert with hormones produced elsewhere, are involved in ongoing male or female physical development.[60]

5.2. It is often assumed that hormones are the driver of all physical sex differences downstream of gonad differentiation.[61] However, analysis of sex-specific genetic architecture in adults reveals some 6500 differences in gene expression, likely to influence development and function outside of hormone effects.[62] Indeed, that "every cell has a sex" dependent on genetics and independent of hormones is recognised and increasingly of scientific interest.[63] REF IOC paper analysis

5.3. A key hormone generating physical differences between males and females is testosterone. Males are exposed to testosterone at three stages of development: 1. in utero; 2. in the post-natal 'minipuberty' period; and, 3. during classic puberty (Figure 1, solid line[64]). Thus, there is an ongoing pattern of differential exposure to testosterone during the development of males and females.

Figure 1. "The three endocrine puberties in boys."
From Becker and Hesse (2020), with permission from S. Karger AG, Basel, CHE



5.4. In utero, testosterone and derived dihydrotestosterone (DHT) are involved in the development of male reproductive anatomy. Testosterone is primarily produced by the male testes.[65] Testosterone promotes the formation of the vas deferens and other male internal genital structures, while DHT is necessary for the development of the penis and prostate gland.[66] The effect of testosterone on somatic development in utero does not appear to be meaningful, and sex differences in fetal size appear unrelated to hormones

[59] Sekido and Lovell-Badge, 2013. Genetic control of testis development. Sexual Development 7:21-32.
[60] Nussey and Whitehead, 2001. Endocrinology: An Integrated Approach. BIOS Scientific Publishers, Oxford, UK.
[61] Lovell-Badge, 1993. Sex determining gene expression during embryogenesis. Philosophical Transactions of The Royal Society (Biological Sciences) 339: 159-164.
[62] Gershoni and Pietrokovski, 2017. The landscape of sex-differential transcriptome and its consequent selection in human adults. BMC Biology 15(1): 7.
[63] For example: Shah et al., 2014. Do you know the sex of your cells? American Journal of Physiology (Cell Physiology) 306(1): C3-C18; Ainsworth, 2017. Sex and the single cell. Nature 550: S6-S8.
[64] Becker and Hesse, 2020. Minipuberty: Why Does it Happen? Hormone Research in Paediatrics 93(2): 76-84.
[65] Richmond and Rogol, 2007. Male pubertal development and the role of androgen therapy. Nature Clinical Practice Endocrinology and Metabolism 3(4): 338-344.
[66] Theakston, 2020. Development of the Reproductive System https://teachmeanatomy.info/the-basics/embryology/reproductive-system

but related rather to the sex-specific genetics of maternal-placental interactions with a male fetus, which affect, for example, nutrient exchange.[67]

5.5 In the post-natal minipuberty period between 1 week to 6 months of age, transient activation of the hypothalamic-pituitary-gonadal axis means males are exposed to a corresponding burst of testosterone.[68] This burst of testosterone supports male penis and testes growth,[69] and is associated with higher growth velocity in the first six months of life,[70] higher weight gain, lower acquisition of body fat and lower body mass index.[71] The transient exposure to testosterone in minipuberty is an excellent candidate to explain the well-established structural differences between males and females in childhood described in **Section 4**.

5.6 At puberty, males experience levels of testosterone up to 20 times greater than in females, driving development during the ensuing teenage years of male secondary sex characteristics.[72] The effects of testosterone on male somatic growth during puberty are well-characterised and hardly require repeating here.[73]

[67] Buckberry et al., 2014. Integrative transcriptome meta-analysis reveals widespread sex-biased gene expression at the human fetal–maternal interface. Molecular Human Reproduction 20(8): 810-819.
[68] Lanciotti et al., 2018. Up-To-Date Review About Minipuberty and Overview on Hypothalamic-Pituitary-Gonadal Axis Activation in Fetal and Neonatal Life. Frontiers in Endocrinology 9: 410.
[69] Boas et al., 2006. Postnatal penile length and growth rate correlate to serum testosterone levels: a longitudinal study of 1962 normal boys. European Journal of Endocrinology 154(1): 125-129.
[70] Kiviranta et al., 2016. Transient Postnatal Gonadal Activation and Growth Velocity in Infancy. Pediatrics 138(1): e20153561.
[71] Becker et al., 2015. Hormonal 'minipuberty' influences the somatic development of boys but not of girls up to the age of 6 years. Clinical Endocrinology 83: 694-701.
[72] Handelsman et al., 2018. Circulating Testosterone as the Hormonal Basis of Sex Differences in Athletic Performance. Endocrine Reviews 39(5): 803-829.
[73] Reviewed in, for example: Hiort, 2002. Androgens and puberty. Best Practice and Research Clinical Endocrinology and Metabolism 16(1): 31-41; Richmond and Rogol, 2007. Male pubertal development and the role of androgen therapy. Nature Clinical Practice Endocrinology and Metabolism 3(4): 338-344.

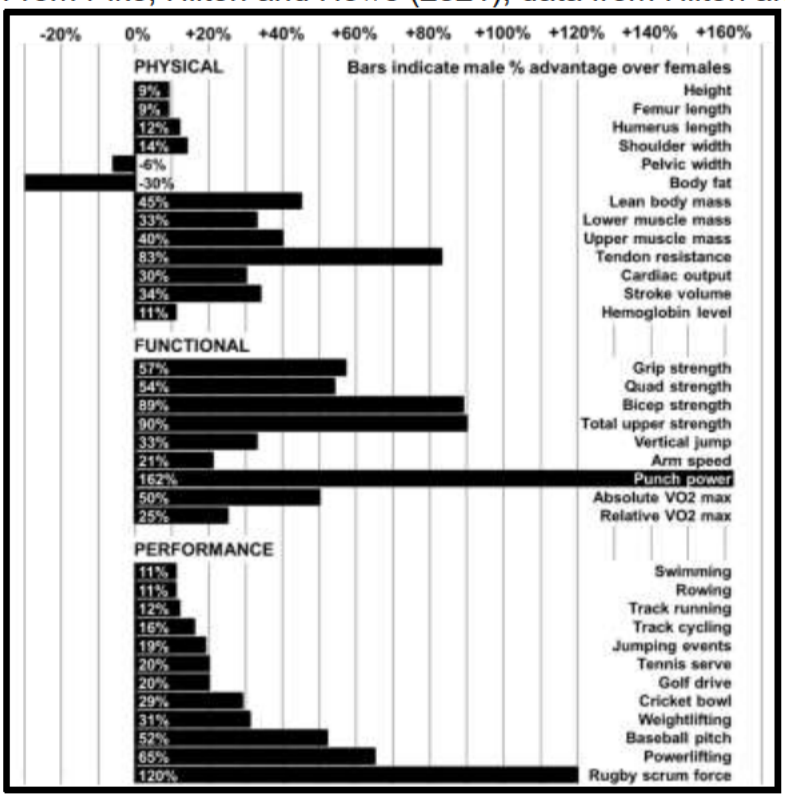
6 Sex and sporting advantage in adolescence and adulthood

6.1 In most athletic sports—those where outcome is affected by speed, stamina, strength and physique—males have a class-level advantage over females. Male advantage is founded in the physical differences, acquired during male development, that underpin functional differences in muscular strength, skeletal levers and proportions, force application, upper to lower body strength, and cardiovascular and respiratory function. In turn, these functional differences confer superior athleticism.[74]

6.2 Examination of sporting records and performances identifies few athletic sporting disciplines where males do not possess performance advantage over females[75], and competitions are typically separated by sex. Volleyball, basketball, soccer and cross-country running are among those where male development provides competitive advantage, and where competitions are therefore separated by sex.

6.3 The physical, functional and performance advantages in adult males are summarised in Figure 2, using reported record performances across multiple sports and sporting actions. Male strength is disproportionately large in the upper body, and sports and sporting movements that require upper body input typically exhibit larger performance gaps than that where lower body strength is key. Performance differences, emerging from the physical and functional differences between adult males and females, have been described as "insurmountable".[76]

Figure 2. Physical, functional and performance differences between males and females.
From Pike, Hilton and Howe (2021); data from Hilton and Lundberg (2021)



[74] For example: Tonnessen et al., 2015. Performance development in adolescent track and field athletes according to age, sex and sport discipline. PLOS One 10(6): e0129014.
[75] For example: Olympic performances https://olympics.com/en/olympic-games/olympic-results; track and field performances https://www.worldathletics.org/stats-zone
[76] Thibault et al., 2010. Women and Men in Sport Performance: The Gender Gap has not Evolved since 1983. Journal of Sports Science and Medicine 9(2): 214-223.

6.4 The significance of male puberty is evidenced by the fact that male performances typically exceed those of elite females in mid-puberty; a comparison of elite female records with male junior records[77] is listed in Table 1. Unsurprisingly, in events like the marathon that are associated with greater strategy and maturity, males are older when they surpass elite female records.

Table 1. Elite female records are surpassed by males in mid-puberty.
Abbreviations: m – metres, km – kilometres, s – seconds, m – minutes, h – hours, yrs – years old

Event	Elite female record	Age at which male records surpass elite female records
100 m	10.49 s	15 yrs (10.20 s)
200 m	21.34 s	14 yrs (20.89 s)
400 m	47.60 s	14 yrs (46.96 s)
800 m	1 m:53.28 s	14 yrs (1 m:51.23 s)
1500 m	3 m:50.07 s	14 yrs (3 m:48.37 s)
5km	14 m:06.62 s	15 yrs (14 m:06.51 s)
10km	29 m:01.03 s	16 yrs (28 m:39.04 s)
Marathon	2 h:17 m:01 s	19 yrs (2 h:11 m:34 s)
High jump	209 cm	14 yrs (217 cm)
Pole vault	506 cm	15 yrs (550 cm)
Long jump	752 cm	15 yrs (785 cm)
Triple jump	1574 cm	15 yrs (1663 cm)
Shot put	2263 cm (4 kg shot)	15 yrs (2386 cm; 5 kg shot)
Discus	7680 cm	15 yrs (7768 cm)
Hammer	8298 cm	14 yrs (8517 cm)
Javelin	7228 cm	14 yrs (7642 cm)

6.5 Importantly, male athletic advantage over females is not limited to those physical and functional differences conferred by male morphology, shape and size. Most obviously, female athletes must typically deal with the effects of the menstrual cycle and the cyclical effects of hormones on training capacity and performance. The menstrual cycle is known to affect cardiovascular, respiratory, brain function, response to ergogenic aids, orthopedics, and metabolic parameters,[78] and represents a barrier to athletic capacity not experienced by males. A third of females report their menstrual flow to be "above average" volume.[79] 37 % of female athletes report heavy menstrual flow, and 90 % report menstrual symptoms, affecting their ability to train and compete.[80]

6.6 Further, injury susceptibility differs between males and females, with subsequent impacts on training time. For example, emerging research shows that compared with males, female rugby players appear more susceptible to concussive injuries, with more severe outcomes. This has been attributed to lower impact resistance in their neck

[77] http://age-records.125mb.com; https://worldathletics.org/records/by-category/world-records
[78] Meignie et al., 2021. The Effects of Menstrual Cycle Phase on Elite Athlete Performance: A Critical and Systematic Review. Frontiers in Physiology 12: 654585.
[79] Bitzer et al., 2013. Women's attitudes towards heavy menstrual bleeding, and their impact on quality of life. Open Access Journal of Contraception 4: 21-8.
[80] Bruinvels et al., 2021. Prevalence and frequency of menstrual cycle symptoms are associated with availability to train and compete: a study of 6812 exercising women recruited using the Strava exercise app. British Journal of Sports Medicine 55: 438-443.

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muscles and more delicate brain structures.[81] A study of sex differences in cultured nerve cells has shown that, compared with male neurons, female neurons have a smaller cross-section and contain fewer, less-dense structural “fibres”; female neurons are more easily damaged when subject to stretch trauma, and they exhibit higher injury responses post-trauma.[82] Female athletes have a higher incidence of anterior cruciate ligament injury than males and poorer response to injury-prevention programmes, well-studied in soccer and typically attributed to female lower body anatomy (hip width, muscle ratio, joint flexibility).[83]

[81] www.rugbypass.com/news/long-term-brain-damage-could-be-a-significantly-bigger-issue-in-womens-rugby-than-mens-says-lead-concussion-doctor/

[82] Dollé et al., 2018. Newfound sex differences in axonal structure underlie differential outcomes from in vitro traumatic axonal injury. Exp Neurol 300:121-134.

[83] Crossley et al., 2020. Making football safer for women: a systematic review and meta-analysis of injury prevention programmes in 11 773 female football (soccer) players. British Journal of Sports Medicine 54: 1089-1098.

7 Sex and sporting advantage in childhood

7.1 While few deny the athletic sporting differences between males and females in adolescence and adulthood, sporting performance gaps between the sexes before puberty are less well-characterised.

7.2 In **Section 4**, I outlined known physical differences between males and females in utero and during childhood. At the level of function leading to athletic performance, large cohort studies of fitness data in typical schoolchildren reveals differences evident from as young as 6 years old. In these childhood fitness programs, females consistently outperform males in the sit and reach test, a measure of flexibility. However, males can run 9.8 % faster over short sprints, jump 9.5 % further from a standing start, complete 33 % more push ups in 30 seconds, complete 16.6 % more shuttle runs in a given time and have 13. 8 % higher grip strength.[84] Young males of 6-7 years old have higher absolute (+11 %) and relative (+8 %) VO_{2max} than female peers.[85]

7.3 The Presidential Fitness Test was a US fitness testing program conducted in middle school and high schools until 2013. Awards were given to schoolchildren in the top 15th percentile in their cohort. I calculated the % difference between the top 15th percentile in male and female schoolchildren aged 6-16 years old, listed in Table 2.[86]

Table 2. Male advantage (%) at the top 15th percentile in the US Presidential Fitness Test for schoolchildren.

Abbreviations: yrs – years old, n – number, s – seconds, cm - centimetres

Age	Curl ups n	Shuttle run s	Sit and reach cm	1 mile s	Pull ups n
6 yrs	3.1	2.4	-36.4	9.6	0.0
7 yrs	5.9	5.0	-30.0	11.6	100.0
8 yrs	5.3	5.9	-33.3	12.3	150.0
9 yrs	5.1	1.8	-45.5	10.4	150.0
10 yrs	12.5	4.6	-33.3	14.7	100.0
11 yrs	11.9	4.8	-38.5	16.6	100.0
12 yrs	11.1	5.8	-42.9	14.3	250.0
13 yrs	15.2	6.9	-50.0	16.8	250.0
14 yrs	19.1	9.9	-43.8	19.4	400.0
15 yrs	18.8	10.0	-37.5	22.1	450.0
16 yrs	24.4	13.9	-33.3	26.8	1000.0

7.4 Thus, physical performance differences among schoolchildren are detectable and measurable in school fitness testing programmes. To begin to systematically analyse pre-puberty and early pubertal differences in sports performance between males and females, I interrogated the extensive track and field performance data available in young people. Track and field events comprise the simple "building blocks'—running, jumping and throwing—that are key to athletic performance in many individual and team sports, including volleyball, soccer and basketball. Thus, track and field event performances can be used to understand likely performance differences in more complex sports.

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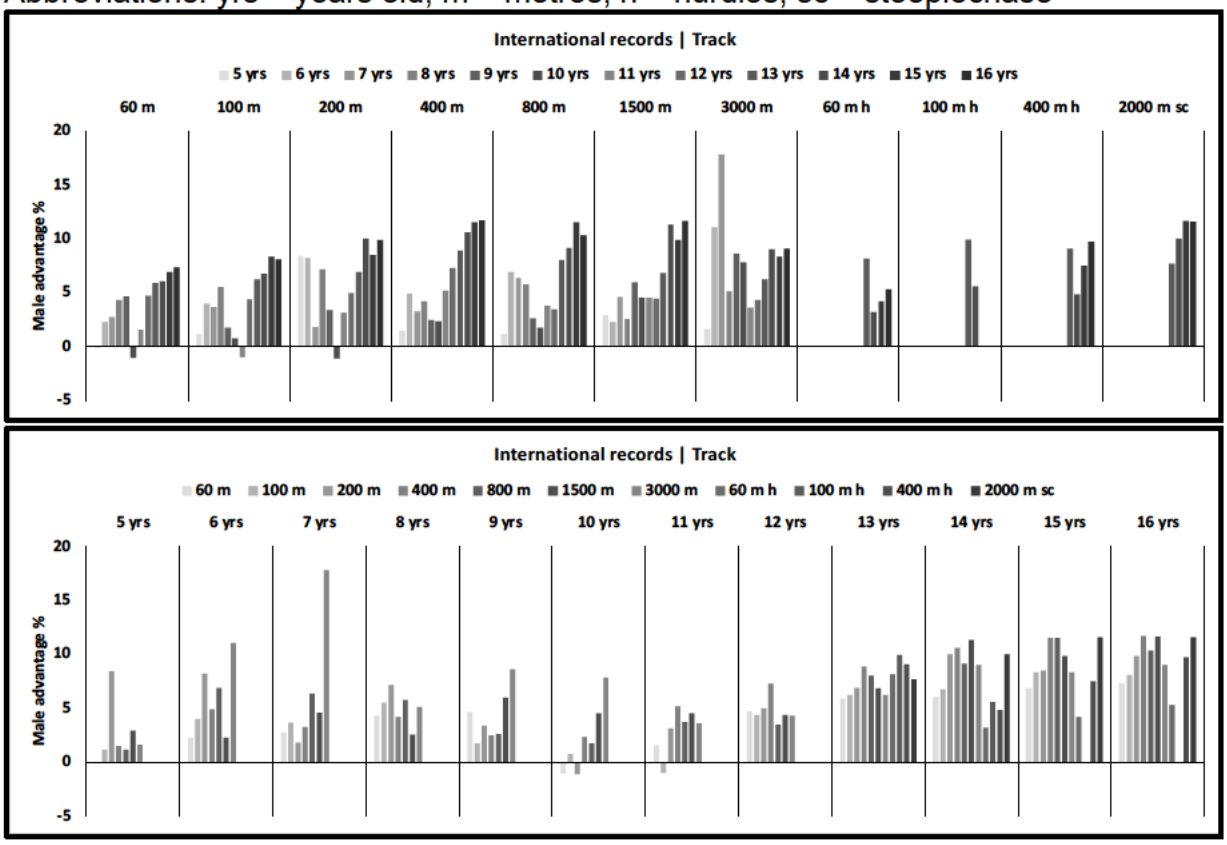
[84] For example: Catley and Tomkinson, 2013. Normative health-related fitness values for children: analysis of 85347 test results on 9–17-year-old Australians since 1985. British Journal of Sports Medicine 47(2): 98–108; Tambalis et al., 2016. Physical fitness normative values for 6–18-year-old Greek boys and girls, using the empirical distribution and the lambda, mu, and sigma statistical method. European Journal of Sport Science 16(6): 736-746.

[85] Eiberg et al., 2005. Maximum oxygen uptake and objectively measured physical activity in Danish children 6–7 years of age: the Copenhagen school child intervention study. British Journal of Sports Medicine 39(10): 725-730.

[86] https://gilmore.gvsd.us/documents/Info/Forms/Teacher%20Forms/Presidentialchallengetest.pdf

7.5 I collected international records in multiple track and field events from both males and females from the ages of 5-16 years old.[87] I then calculated the % difference between the male record and equivalent female record. The male advantages (%) in track, stratified by both event (upper panel) and age (lower panel), are shown in Figure 3. In track events, male advantage is clear in all age groups and for all events.

Figure 3. The male advantage over females in international schoolchildren records in track events, stratified by event (upper panel) and age group (lower panel). Abbreviations: yrs – years old, m – metres, h – hurdles, sc – steeplechase



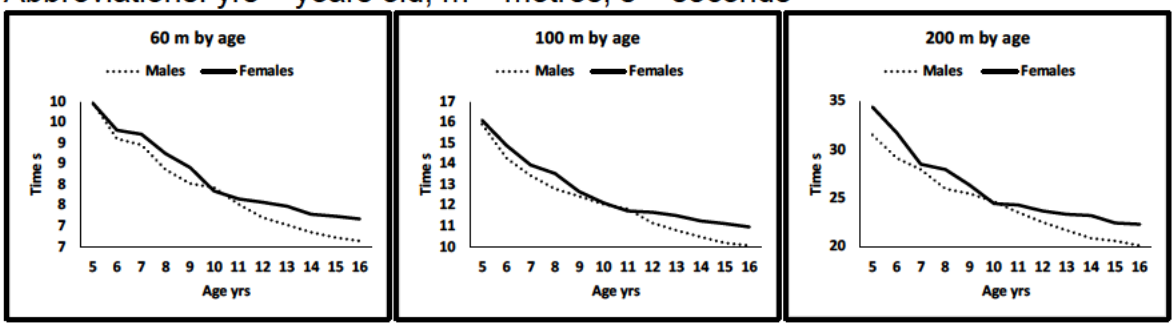
7.6 There are four track events where female schoolchildren appear to outperform their male peers, listed in Table 3. I examined the age progression of these events to seek to understand this apparent female advantage. These data are shown in Figure 4. For 60 m at 5 years old, in the absence of a preceding datapoint, it is impossible to evaluate the female advantage here. For 60 m at 10 years old, the male record appears slightly slower than predicted, with no specific explanation for this beyond typical variation. In this same event, the female record is faster than expected, possibly explained by earlier onset of puberty and associated growth spurt that provides transient 'catch up' with male peers. For 100 m at 11 years old and 200 m at 10 years old, again the female records appear faster than expected, again likely underpinned by pubertal growth spurt in these female athletes.

[87] International age records http://age-records.125mb.com

Table 3. Female advantage in international schoolchildren track records.
Abbreviations: yrs – years old, m – metres

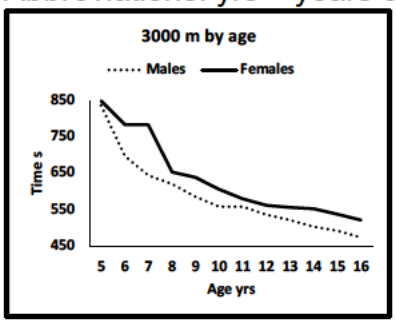
Event	Age group	Female advantage %
60 m	5 yrs	0.1 %
	10 yrs	1.0 %
100 m	11 yrs	0.9 %
200 m	10 yrs	1.1 %

Figure 4. Age progression in the 60 m, 100 m and 200 m sprints in international schoolchildren records.
Abbreviations: yrs – years old, m – metres, s – seconds



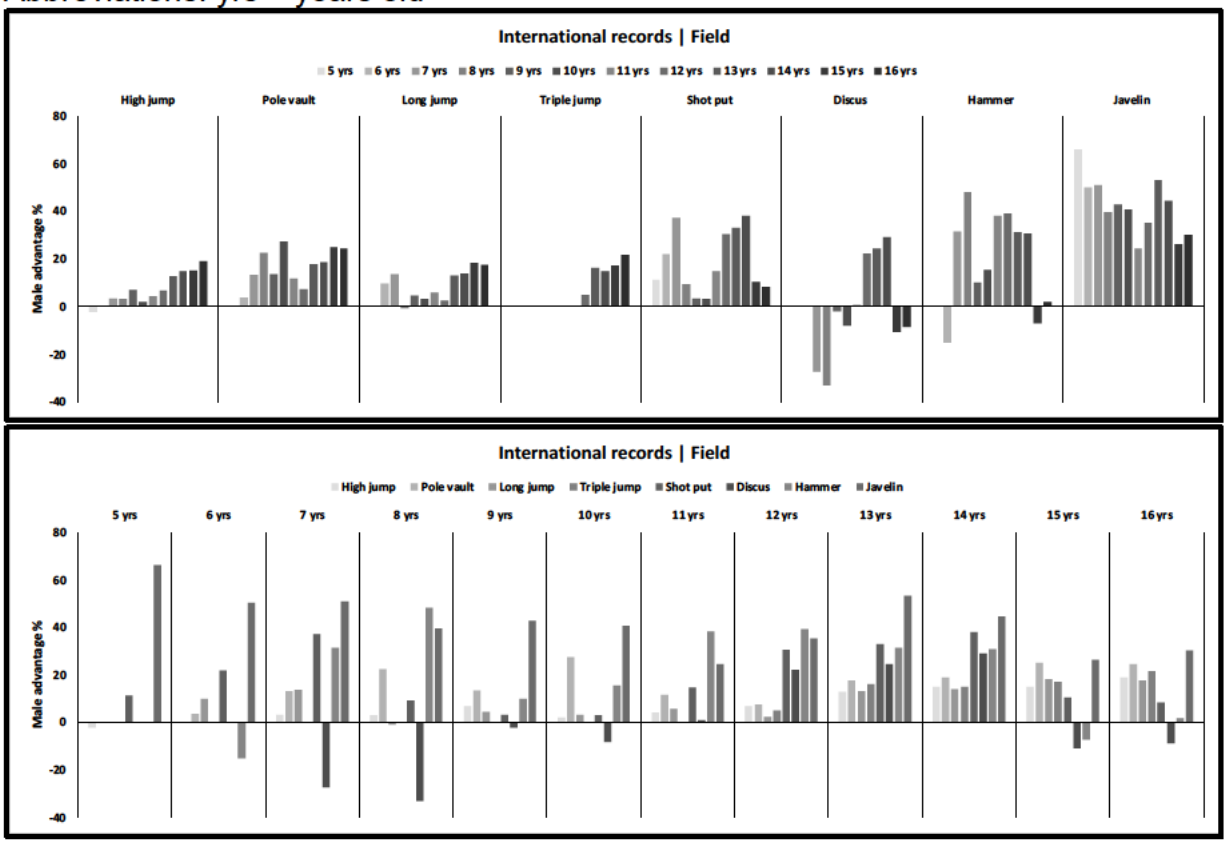
7.7 Also evident in this dataset is an unusually large male advantage for 3000 m at 7 years old. Analysis of the age progression for this event, shown in Figure 5, reveals this is underpinned by an unexpectedly poor female record for 3000 m at 7 years old. Thus, the extent of male advantage here is likely an overestimate of the true performance gap.

Figure 5. Age progression in the 3000 m in international schoolchildren records.
Abbreviations: yrs – years old, s – seconds



7.8 The male advantages (%) in field events, stratified by both event (upper panel) and age (lower panel), are shown in Figure 6. In field events, male advantage is again evident in all age groups and for all events, although this appears less systematic than in track events.

Figure 6. The male advantage over females in international schoolchildren records in field events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old

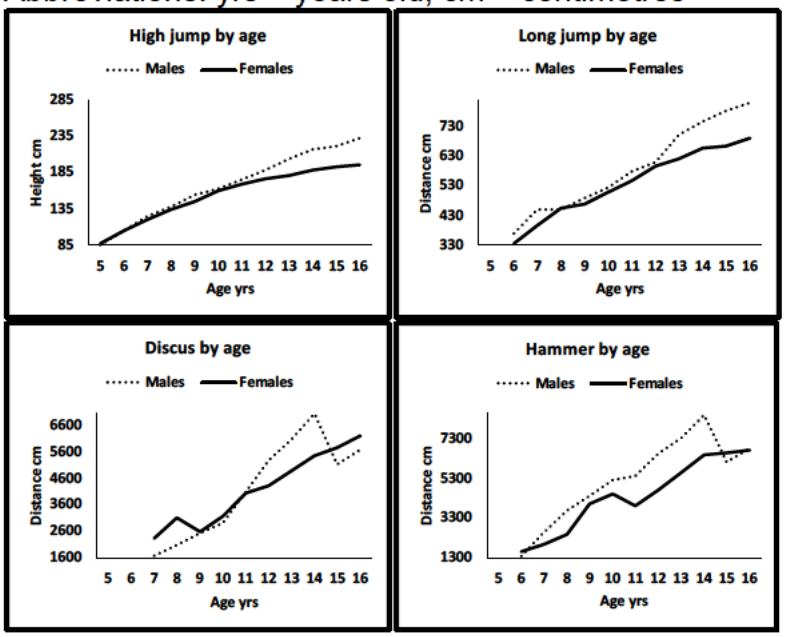


7.9 There are several field events where female schoolchildren appear to outperform their male peers, listed in Table 4. I examined the age progression of these events to seek to understand this apparent female advantage. These data are shown in Figure 7. For the high jump at 5 years old, in the absence of a preceding datapoint, it is impossible to evaluate the female advantage here. For the long jump at 8 years old, the female advantage appears to be explained by the convergence of an unusually poor male record and unusually good female record in this event.

Table 4. Female advantage in international schoolchildren field records.
Abbreviations: yrs – years old, m – metres

Event	Age group	Female advantage %
High jump	5 yrs	2.3 %
Long jump	8 yrs	0.9 %
Discus	7 yrs	27.4 %
	8 yrs	33.1 %
	9 yrs	2.1 %
	10 yrs	8.1 %
	15 yrs	10.8 %
	16 yrs	8.7 %
Hammer	6 yrs	15.1 %
	15 yrs	7.2 %

Figure 7. Age progression in the high jump, long jump discus and hammer in international schoolchildren records.
Abbreviations: yrs – years old, cm – centimetres



7.10 There are several throw events where female schoolchildren appear to outperform their male peers by a large distance. However, there are important confounding factors in throwing events, given that the weight of throwing implements can differ between male and female athletes at different ages. For the discus, girls at 7-8 years old throw a discus weighing 500 g, compared to boys of the same age using a 750 g discus. I hypothesise that a similar implement weight at 7-8 years old would mitigate or remove the apparent female advantage here. Between 9-14 years old, both sexes use a 1 kg discus. Performance between males and females seems broadly matched until 11 years old, which may be underpinned by earlier female puberty. Males open up the performance gap at 11 years old. At 15 years old, boys switch to a 2 kg discus. I believe it is reasonable, given the increasing male gap to 14 years old with the same implement weight of 1 kg, that a matched implement weight between males and females at 15-16 years old would reverse the apparent female advantage in favour of clear male advantage.

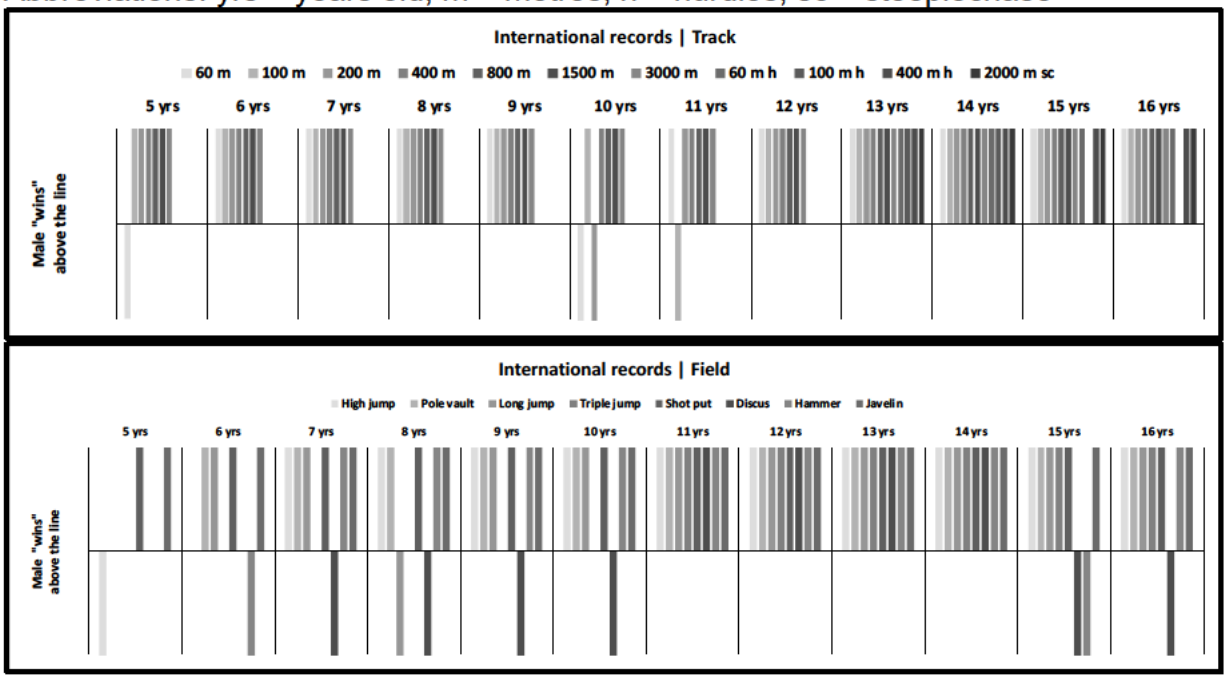
7.11 For the hammer, male and females use a 2 kg implement between the ages of 6-10 years old. At 6 years old, in the absence of a preceding datapoint, it is impossible to evaluate the female advantage here. Male advantage is evident from 7-10 years old; the 'catch up' with male peers at ages 9-10 years old may be explained by the physical changes of female puberty. Between the ages of 11-14 years old, both males and females use a 4 kg hammer, and male advantage is consistent through these ages. At 15 years old, males switch to a 7.26 kg hammer. I believe it is reasonable, given the male advantage evident throughout the time period where both sexes use a 4 kg hammer, that a matched implement weight between males and females at 15-16 years old would reverse the apparent female advantage in favour of clear male advantage.

7.12 Interestingly, male advantage is evident in all shot put and javelin events at all ages, despite increases in implement weight at 15-16 years old for males.

7.13 I formulated a null hypothesis: if there are no sex differences in athletic performances in schoolchildren, males and females are equally likely to hold the best record in any event. Therefore the frequency of males with the best record should be approximately equal to

the frequency of females with the best record. To interrogate this statistically, I scored all track and field events at all ages as a binary variable of male "wins" versus female "wins" (whichever record was the fastest, longest, etc). I ignored potential confounding explanations in various events; that is, female advantage was scored as a "female win", even if the female advantage is likely an artifact of, for example, earlier puberty or lighter implement weight. Thus, this scoring is deliberately generous to ensure the strength of any findings. Scoring data are visualised in Figure 8, with track events in the upper panel and field events in the lower panel. It is already clear from this analysis that the majority of "wins" go to male schoolchildren.

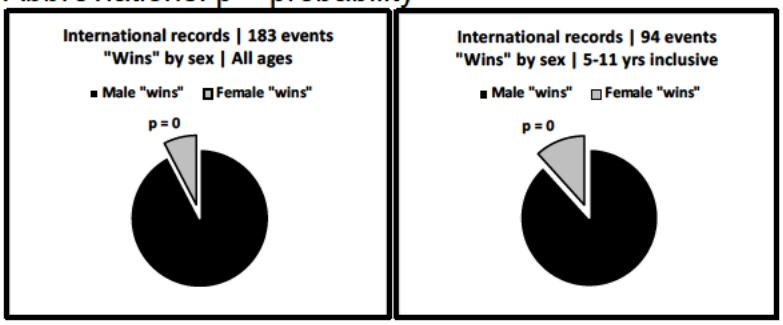
Figure 8. Male versus female "wins" in international schoolchildren records, scored in track events (upper panel) and field events (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, sc – steeplechase



7.14 I counted the frequency of male "wins" versus female "wins" at all ages and in a sub-analysis limited to events in pre-puberty (5-11 years old) age groups. I then calculated the probability that the frequency of male "wins" versus female "wins" would occur by chance.[88] These data are shown in Figure 9. The majority of "wins" go to male schoolchildren, whether across all age groups or limited to pre-puberty age groups. The chances of this frequency of male "wins" occurring by chance in either age grouping is calculated at a probability of effectively zero (p = 0).

[88] https://homepage.divms.uiowa.edu/~mbognar/applets/bin.html

Figure 9. The frequency of male versus female "wins" across pooled events in all age groups (left) and limited to pre-puberty age groups (right).
Abbreviations: p – probability



7.15 Following the same process for international records above, I analysed junior records from 8-16 years old from USA Track and Field (USATF)[89] and the US Amateur Athletics Union (AAU).[90] For brevity here, these datasets are compiled in **Appendix 3** (USATF) and **Appendix 4** (AAU). These national datasets confirm the results obtained from international records. **To summarise the data obtained from international and national schoolchildren records in track and field: 1. male advantage over female peers is evident across track and field events from 8 years old onwards; 2. males systematically outperform their female peers from 8 years old at a frequency that is vanishingly unlikely to result by chance.**

7.16 Again, following the same process for international records above, I analysed Arizona middle school records from 8-16 years old (available to 2014).[91] For brevity here, this dataset is compiled in **Appendix 5**. This dataset confirms that male advantage over female peers is predominant across track and field events from 8 years old. In these state level records, more female "wins" are scored in lower age groups than seen in international and national records. However, the frequency of male "wins" between 8-12 yrs old is still statistically unlikely to result from chance ($p = 0.043$, where $p = 0.05$ is the "significance" threshold).

7.17 I analysed the outcomes of two individual middle-school competitions. The first was the Kyrene District Track and Field Championship, held in April 2023.[92] Middle-schoolers participated in 13 events, and I calculated the male advantage for the winners of each matched event. These data are shown in Figure 10. In this school district championship, male advantage was evident in all events. I pooled all events then plotted the frequency of male versus female "wins" in this group of athletes. Again, I calculated the probability that the male "win" frequency would occur by chance. These data are shown in Figure 11. The probability that males would win all these events by chance is vanishingly low.

[89] https://www.usatf.org/resources/statistics/records/championship-meet-records/usatf-national-junior-olympic-track-field-champion
[90] https://aautrackandfield.org/Results
[91] http://www.usatf.com/assoc/az/records.html
[92] https://www.athletic.net/TrackAndField/meet/486419/results/all

Figure 10. The male advantage over females at the Kyrene District Track and Field Championship, held in April 2023.
Abbreviations: m – metres, h – hurdles, SMR – sprint medley relay

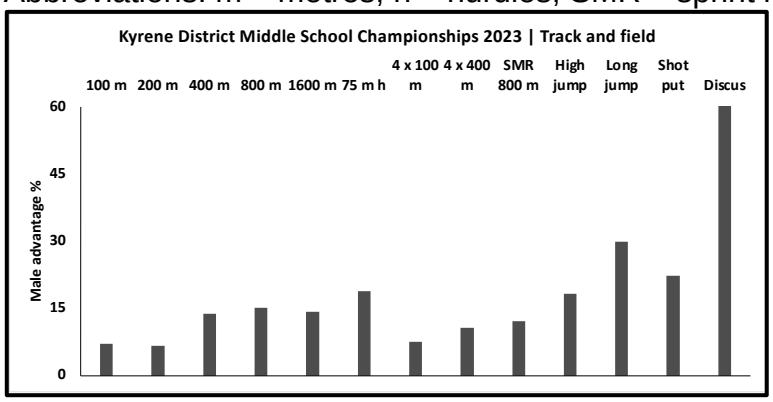
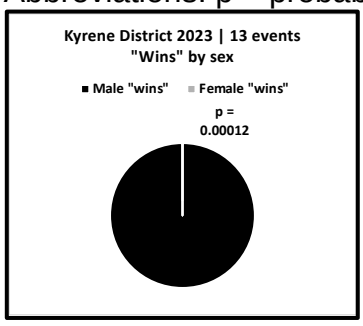
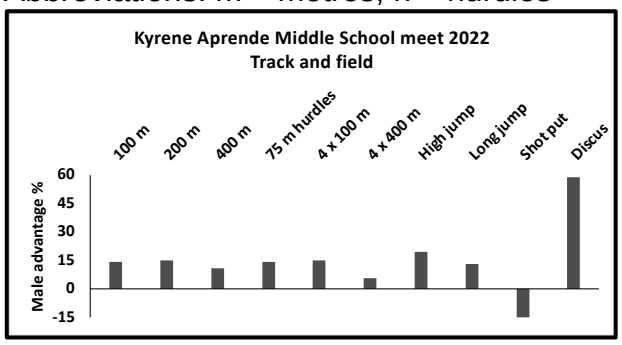


Figure 11. The frequency of male versus female "wins" across the pool of events at the Kyrene District Track and Field Championship, held in April 2023.
Abbreviations: p – probability



7.18 The second middle-school competition I analysed was the Kyrene Aprende Middle School Track and Field meet, held in July 2022.[93] Middle-schoolers participated in 12 events; however, the girls' times for the 800 m and 1600 m were not recorded on the scoresheets so I was unable to include these in my analysis. I calculated the male advantage for the matched winners in the remaining 10 events. These data are shown in Figure 12. In this single school athletics meet, male advantage was evident in all events except the shot put, where the apparent female advantage was an unexpectedly large 14.8 %.

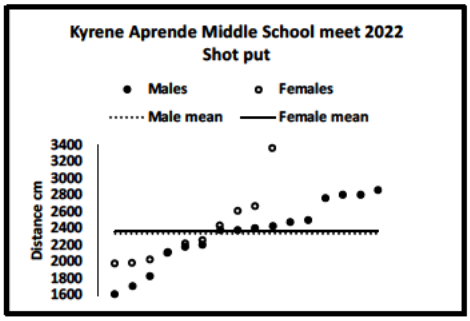
Figure 12. The male advantage over females at the Kyrene Aprende Middle School Track and Field meet, held in July 2022.
Abbreviations: m – metres, h – hurdles



[93] https://www.kyrene.org/Page/55102

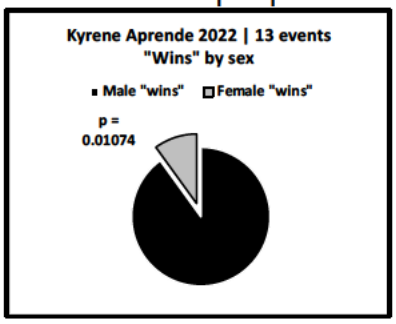
7.19 To understand the source of this female advantage in shot put, I analysed the puts of all the males and females at this middle school meet. These data are shown (with puts in increasing order of distance achieved) in Figure 13. The winner of the female competition putted 3360 cm, well beyond the second placed girl at 2670 cm. This winning female performance is 4.2 standard deviations from the female mean put distance, indicating an extraordinary performance with odds of occurrence of approximately 1 in 15000. A comparison of the mean distance putted by boys and girls shows them to be quite similar; however, the female winner is skewing this mean distance by 110 cm (the male winner only skews the male mean by 35 cm).

Figure 13. Analysis of puts at the Kyrene Aprende Middle School Track and Field meet, held in July 2022.
Abbreviations: cm - centimetres



7.20 I pooled all events then plotted the frequency of male versus female "wins" in this group of athletes. Again, I calculated the probability that the male "win" frequency would occur by chance. These data are shown in Figure 14. The probability that males would win almost all the events by chance is very low (p = 0.05 is the "significance" threshold).

Figure 14. The frequency of male versus female "wins" across the pool of events at the Kyrene Aprende Middle School Track and Field meet, held in July 2022.
Abbreviations: p – probability



7.21 Analyses of international, national and state track and field performances in male and female schoolchildren evidence sex differences in athletic performance, even before puberty. Sex differences in athletic performance are evident in middle school track and field meets. Collectively, these data demonstrate that female children require a female category of sport to win.

7.22 Childhood male athletic advantage over females has been proposed as social in origin. That is, higher engagement in sport and exposure to rougher play may represent 'training advantage' over females who are somewhat socialised to engage in less

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physical activity.[94] However, despite suggesting that childhood performance gaps are possibly social in origin, Thomas and French (1985) identify an extremely large gap in throwing differences, evident from age 3 years old, that are “unlikely to be completely environmentally caused” and are unlikely, based on biological factors, to be eliminated by training. The performance gap in international and national track and field records, evident even before puberty, somewhat controls for this socialisation effect, given that one might expect engaged, sporty girls to be as well-trained as their male peers.

[94] For example: Thomas and French, 1985. Gender differences across age in motor performance a meta-analysis. Psychol Bull 98(2):260-282.

8 Sports categories and concepts of advantage

8.1 Sports where performance or competitor safety is affected by sex routinely employ a protected female category that excludes males, to secure fairness for (and, discipline-dependent, safety of) female athletes. This separation on the basis of sex in pursuit of fair, safe sports and sporting opportunities for female athletes is permissible under much national equality legislation, including, for example, the UK Equality Act 2010.[95]

8.2 Misunderstandings regarding the nature of categories and advantage are common. Sports categories control for baseline physiological differences in sex, age, and impairment (and occasionally weight) that affect results or outcomes independently of the characteristics sporting competition seeks to reward: talent, strategy, training and dedication. Various initiatives like leagues, which operate alongside categories, exist to permit participation of those with different amounts of talent, strategy, training and dedication.

8.3 Categories are rationalised on biological principles, understanding what effect factors like sex and age have on the human body. They exist to ensure physiological “bonuses” (being male, being young) do not obscure outcomes that should depend on talent, strategy, training, and dedication. It is via categories that fairness is achieved, and we ensure that winning opportunities for the more talented athlete—a fundamental characteristic of sport—are preserved. Protected categories like the female category are a necessary inclusion measure to ensure females have an equal opportunity to compete in sports.

8.4 Advantage exists regardless of magnitude. Indeed, sports bodies have a history of regulating for even very small advantages. For example, inside lane track runners closer to the traditional start gun hear the gun more quickly and more loudly than those in outside lanes, offering them a small kind of advantage unavailable to the whole field. To combat this advantage, worth around 150 milliseconds in a staggered start of a 400m track, runners typically now start races via a loudspeaker at each block.[96] Even if an apparent advantage is small, a category or rule operates to exclude any quantity of it.

8.5 A common argument is to frame ‘advantage’ as simply a property of results (for example, any person who is faster than any other has ‘advantage’, while people who are equally fast are said to be fairly-matched), one undermines the very existence of categories. The logical outcome is sports organised not to reward talent but to reward a combination of talent and talent-independent physical properties that together deliver a winning outcome. In such a framework, almost all sports at every competitive level will be dominated by able-bodied males aged around 20-35 years old.

8.6 What has traditionally been described as a “girl’s/women’s category” is more precisely understood as a category for females that excludes males who have acquired any magnitude of male athletic advantage by virtue of biology, regardless of performance relative to the female field. The ineligibility of those with any male advantage is necessary to maintain the integrity of the female sports category.

8.7 Puberty, where we see a sharp divergence of male and female athletic performance, is typically regarded as the age at which a protected female category becomes necessary. I believe, given the evidence I have presented in **Section 7** that demonstrates male advantage in childhood, that is justified from pre-puberty ages to institute a protected female category that excludes any male advantage, should fairness for young female athletes be a priority for regulators.

[95] UK Equality Act 2010, Part 14, Section 195.
[96] Holmes, 2008. Olympic start gun gives inside runners an edge. New Scientist, 23rd June 2008.

9 Treatment of transgender girls and transgender women

9.1 Transgender girls and transgender women may take social, pharmaceutical and/or surgical steps to be perceived and treated as if they were female. In adulthood, transgender women may opt for testosterone suppression (for example, via gonadotropin-releasing hormone [GnRH] agonists, spironolactone or cyproterone acetate) then/or surgical removal of the testes; both of these interventions have the effect of lowering testosterone levels to those of females[97] and reducing the functional or visual impact of male physical characteristics. Estrogen supplementation typically promotes feminisation of, for example, breast tissue.[98]

9.2 Early pharmaceutical interventions in transgender girls may involve blocking male puberty via GnRH agonists ("puberty blockers"), administered after the onset of puberty (at least Tanner stage 2; in male children, the appearance of pubic hair, increase in testicular volume and reddening of scrotum skin).[99] This is typically followed by a regime of cross-sex hormones from 16 years old.

9.3 Many children reporting gender dysphoria or incongruent gender identity desist; that is, gender identity issues resolve with puberty.[100] For this reason, puberty blockers are not administered until after the onset of puberty and there is observed demonstrable persistence of gender identity issues. Furthermore, the reported effects and side-effects of puberty blockers are serious, including long-term effects on bone growth, brain development, fertility and sexual function, and short-term effects like headaches, hot flashes, mood swings, and depression and anxiety,[101] necessitating caution with their prescription.

9.4 Considering the potential for medical harm while outcomes remain uncertain, many jurisdictions have cautioned against or restricted the use of puberty blockers in children, including the Swedish National Board of Health and Welfare,[102] the Finnish Health Authority,[103] the French National Academy of Medicine[104] and the Norwegian Healthcare Investigation Board.[105] The UK NHS has recently restricted puberty blockers within clnical research.[106] Pioneers of the original protocol for treatment of childhood dysphoria have advocated re-evaluation considering the rapidly-changing cohort demographics.[107]

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[97] Nishiyama, 2014. Serum testosterone levels after medical or surgical androgen deprivation: a comprehensive review of the literature. Urologic Oncology 32(1): 38.e17-28.
[98] Unger, 2016. Hormone therapy for transgender patients. Translational Andrology and Urology. 5(6): 877-884.
[99] Puberty progression is assessed using "Tanner staging", which describes the typical physical changes in boys and girls using landmarks of external genitalia in males (testicular volume, penis length and skin appearance), quantity and coarseness of pubic hair in both sexes, and breast development in girls. In males, Tanner stage 2 indicates the first signs of puberty, around the age of 11 years old, comprising the appearance of downy pubic hair, an increase in testicular volume and reddening of the scrotum skin. At Tanner stage 3, around the age of 13 years old, the penis begins to grow in length. Testicular volume increase and penis growth continues during later stages, and pubic hair becomes course and curly. For more information, see: https://childgrowthfoundation.org/wp-content/uploads/2020/03/Puberty-and-Tanner-Stages_v2.0.pdf
[100] Wallien and Cohen-Kettanis, 2008. Psychosexual outcome of gender-dysphoric children. Journal of the American Academy of Child and Adolescent Psychiatry 47(12): 1413-1423.
[101] Reported by various healthcare providers, for example: Mayo Clinic, NHS, St. Louis Children's Hospital.
[102] https://www.socialstyrelsen.se/globalassets/sharepoint-dokument/artikelkatalog/kunskapsstod/2022-3-7799.pdf
[103] https://palveluvalikoima.fi/documents/1237350/22895838/Summary+transgender.pdf/2cc3f053-2e34-39ce-4e21-becd685b3044/Summary+transgender.pdf?t=1592318543000
[104] https://segm.org/sites/default/files/22.2.25-Communique-PCRA-19-Medecine-et-transidentite-genre.pdf
[105] https://www.bmj.com/content/bmj/380/bmj.p697.full.pdf
[106] https://www.england.nhs.uk/wp-content/uploads/2023/06/Interim-service-specification-for-Specialist-Gender-Incongruence-Services-for-Children-and-Young-People.pdf
[107] de Vries, 2020. Challenges in Timing Puberty Suppression for Gender-Nonconforming Adolescents. Pediatrics 146(4): e2020010611.

9.5 When prescribed as above, puberty blockers do not, by definition, block the entirety of male puberty. They do not block any hormone-derived pre-puberty effects on male development. They are unlikely to interfere with genetic effects on male development.

10 Transgender women in sport

10.1 Given the role of testosterone in the development of the male characteristics that matter for sporting performance, and bearing in mind the typical pharmaceutical and medical treatment sought by transgender girls and transgender women, the International Olympic Committee (IOC) and other sporting federations have historically sought to include transgender women in female sports by regulating levels of testosterone prior to inclusion in female competition.[108] More recently, the IOC have suggested that "*testosterone levels could be investigated as a means to mitigate performance*" in transgender women.[109] It is inferred that the IOC believe testosterone suppression may be sufficient to remove the male performance advantage provided by male-typical secondary sex characteristics.

10.2 In 2020, with the IOC equivocating over a review of their testosterone guidelines, Dr Tommy Lundberg and I tested the existing guidelines' promise to protect fair competition, by reviewing peer-reviewed published longitudinal changes in muscular and skeletal metrics in transgender women suppressing testosterone in adulthood for a minimum of 12 months.[110] Having reviewed measures of bone density, lean body mass, muscle mass and strength tests, we identified a unified consensus in original studies covering approximately 800 transgender women that skeletal metrics like height and bone length were unaffected, bone mass was preserved, and muscle mass and strength was decreased by 4% over 12 months of testosterone suppression. Within this dataset, compared with female control cohorts, higher muscle mass/strength values—between +13-41 %—were maintained for at least three years after testosterone suppression (the limit of current longitudinal studies).

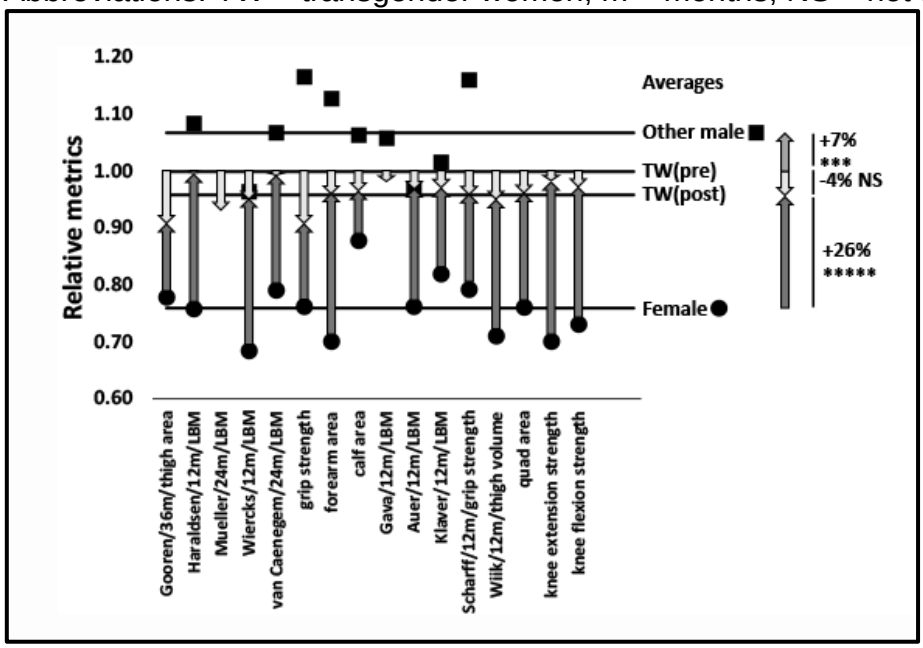
10.3 These observations were subsequently reinforced by a systematic review of the same dataset published by another group later in 2021, which concluded that, in transgender women, "*hormone therapy decreases strength, [lean body mass] and muscle area, yet values remain above that observed in cisgender women, even after 36 months. These findings suggest that strength may be well preserved in transwomen during the first 3 years of hormone therapy.*"[111]

10.4 To gain an overall picture of the baseline metrics and effects on muscle mass and strength in transgender women pre- and post- at least 12 months of testosterone suppression, I compared pre- and post- metrics for transgender women across the Hilton and Lundberg dataset with data from control males and females, shown in Figure 15. Original study metrics were converted to relative percentages, with pre-suppression metrics in transgender women set at 100%. The 4% reduction in muscle mass and strength in transgender women pre- and post- at least 12 months of testosterone suppression was not statistically significant. The difference between transgender women and control males was statistically significant, with transgender women pre- and post- at least 12 months of testosterone suppression deviating from control males by -7% and -11%, respectively. The difference between transgender women and females is also statistically significant; transgender women pre- and post- at least 12 months of

[108] https://stillmed.olympic.org/Documents/Commissions_PDFfiles/Medical_commission/2015-11_ioc_consensus_meeting_on_sex_reassignment_and_hyperandrogenism-en.pdf
[109] Martowicz et al., 2023. Position statement: IOC framework on fairness, inclusion and non-discrimination on the basis of gender identity and sex variations. Br J Sports Med 57:26–32.
[110] Hilton and Lundberg, 2021. Transgender Women in the Female Category of Sport: Perspectives on Testosterone Suppression and Performance Advantage. Sports Medicine 51, 199–214. Note: the date disparity of the published paper represents the gap between article submission and publication.
[111] Harper et al., 2021. How does hormone transition in transgender women change body composition, muscle strength and haemoglobin? Systematic review with a focus on the implications for sport participation. Br J Sports Med 55: 865-872.

testosterone suppression deviate from control females by +35% and +30%, respectively. It appears that for metrics of muscle mass and strength, transgender women remain within 'male range'.

Figure 15. Relative metrics in transgender women pre- and post- testosterone suppression, compared with control males and females.
Abbreviations: TW – transgender women, m – months, NS – not significant



10.5 In addition to the longitudinal data captured by the above reviews, there are three significant cross-sectional studies of physical metrics in transgender women suppressing testosterone. The first found that transgender women, after an average of 8 years of suppressed testosterone, had a lean body mass in the 90th percentile for females, and grip strength that remained 25 % higher than the female reference value.[112] The second, in transgender women suppressing testosterone for just over 3 years, showed that those transgender women had a mean lean body mass 18 % higher than the mean in control females.[113] The third found that transgender women suppressing testosterone for over 14 years retained higher cardiopulmonary capacity metrics and higher hand grip strength than female controls.[114]

10.6 In 2015, to assess sports performance in transgender women, an observational cohort study of transgender women runners was performed, studying race times before and after testosterone suppression.[115] Participants were club-level middle-distance runners. After applying an age-grading formula typically reserved for Masters athletes, performance in the female category was judged to be maintained at a level equivalent to pre-suppression performance in the male category. This study had a sample size of eight runners self-reporting times that were unverifiable in 50% of cases and spanning a period of decades. The study could not make any controls for ageing, training, diet,

[112] Lapauw et al., 2008. Body composition, volumetric and areal bone parameters in male-to-female transsexual persons. Bone. 43(6):1016–1021.
[113] Bretherton et al., 2021. Insulin resistance in transgender individuals correlates with android fat mass. *Ther Adv* Endocrinol Metab 12:2042018820985681.
[114] Alvares et al., 2022. Cardiopulmonary capacity and muscle strength in transgender women on long-term gender-affirming hormone therapy: A cross-sectional study. Br J Sports Med 56: 1292-1298.
[115] Harper, 2015. Race times for transgender athletes. Journal of Sporting Cultures and Identities 6:1-9.

injury, running course, or course weather conditions. The overall cohort analysis included times from runners who had experienced chronic injury and loss of fitness, resulting in poorer-than-expected performance within the female field. However, excluded from the overall analysis was a runner who had achieved a far higher ranking competing in female running than in male running. This individual improved ranking significantly, and even recorded a marathon that was faster than previous marathon performance in the male category, but was considered an outlier who had seriously intensified her training after transition into female sport. This individual demonstrates, as argued in Hilton and Lundberg, that training during testosterone suppression can mitigate negative performance effects.

10.7 There have been two studies of athletic performance in military personnel using basic fitness testing data.[116] While not athletes, these individuals do represent a trained population of transgender people. Both studies tracked changes in push-up, sit-up and 1.5 mile run performance during annual fitness testing over 3 or 4 years of testosterone suppression. Such tests are 'work to target': recruits are aware of targets that must be achieved to pass the fitness testing process, minimum performances must be achieved for each test, and a cumulative score threshold must be reached to pass the fitness test. Individual officers have the latitude to "choose" how their scores are allocated, such that a particularly strong runner has a lower need to gain points during the push-up test (for example). The performances cannot thus be assessed as maximal performances, but instead may be considered as paced performances with conscious knowledge of a required standard. The authors of the first study acknowledge that, despite being in a controlled environment of the Air Force, the exercise intentions and training habits of the recruits was unknown, and over a period of three years, changes in training with material implications for muscle and cardiovascular performance cannot be known.

10.8 Significantly, the data from the two studies of athletic performance in military personnel make contradictory findings, presented in Table 5. Roberts et al. (2021) finds that both push-up and sit-up performance are statistically equivalent to female performance after 2 years while advantage in running performance is retained to 2 years. However, Chiccarelli et al. (2022) finds that push-up advantage is retained beyond 4 years, sit-up performance is statistically equivalent to female performance at 4 years and running performance is statistically equivalent to female performance at 2 years.

10.9 This set of performance studies suffer from small numbers of participants, lack of controls for performance times, and issues regarding the validity of performance tests. They cannot be used in isolation to inform sports policy, particularly when the overwhelming body of evidence suggests that the effects of testosterone suppression on important metrics like muscle mass and strength are marginal and that male development, and thus male advantage, cannot be reversed.

[116] Roberts et al., 2021. Effect of gender affirming hormones on athletic performance in transwomen and transmen: Implications for sporting organisations and legislators. Br J Sports Med 55:577-583; Chiccarelli et al., 2022 Fit transitioning: When can transgender airmen fitness test in their affirmed gender? Mil Med 2022;usac320.

Table 5. A comparison of the findings of two studies of athletic performance in military personnel.

Abbreviations: NA – not applicable, * – year at which statistical parity with females is reached

	Roberts et al., 2021 Year group % change (% advantage over female controls)			**Chiccarelli et al., 2022** Year group % change (% advantage over female controls)		
	Push-ups	**Sit-ups**	**Running**	**Push-ups**	**Sit-ups**	**Running**
Pre-transition	NA (+45.5 %)	NA (+17.3 %)	NA (+17.2 %)	NA (66.3 %)	NA (+28.3 %)	NA (+17.8 %)
Year 0-1	-5.7 % (+37.2 %)	+1.1 % (+18.6 %)	-7.1 % (+11.3 %)	-13.0 % (+44.7 %)	-6.1 % (+20.5 %)	-10.4 % (+9.2 %)
Year 1-2	-3.1 % (+32.9 %)	-4.3 % (+13.6 %)	-4.4 % (+7.5 %)	-9.4 % (+31.0 %)	-2.6 % (+17.3 %)	-4.5 % (+5.1 %)*
Year 2-3	-19.9 % (+6.5 %)*	-13.5 % (-1.8 %)*	+3.3 % (+10.5 %)	-2.0 % (+28.3 %)	-5.2 % (+11.2 %)	-0.0 % (+5.1 %)*
Year 3-4				-8.3 % (+17.7 %)	-2.6 % (+8.3 %)*	-5.2 % (+0.2 %)*

11 Transgender girls in sport

11.1 Most sporting federations exempt from testosterone regulations those who have blocked puberty before cross-sex hormone treatment. To my knowledge, there is no published data on muscle mass and strength metrics in a cohort of transgender girls who have blocked puberty from Tanner stage 2.

11.2 Recently available is a study by Boogers et al. (2022) called, "Trans girls grow tall: adult height is unaffected by GnRH analogue and estradiol treatment."[117] In this study, transgender girls who had received puberty blockers from around 13 years of age, then cross-sex hormones at 16 years of age, acquired an average adult height of 180.1-185.3 cm, far larger than the population female average (170.7cm) and around the population male average (183.8cm). The authors conclude that the driver of height acquisition is genetic in origin, and not a result of testosterone during puberty.

11.3 In two studies where male puberty was partially-blocked, lean body mass in young adulthood remains higher than in reference females[118] and grip strength remains higher than in a matched cohort of transgender boys.[119]

11.4 Claims that transgender girls who block puberty do not acquire any male athletic advantage in terms of skeletal structure and/or muscle mass are speculative.

[117] Boogers et al., 2022. Trans girls grow tall: adult height is unaffected by GnRH analogue and estradiol treatment. Journal of Clinical Endocrinology and Metabolism. Epub ahead of print, PMID: 35666195.
[118] Klaver et al., 2018. Early Hormonal Treatment Affects Body Composition and Body Shape in Young Transgender Adolescents. Journal of Sexual Medicine 15(2): 251-260.
[119] Tack et al., 2018. Proandrogenic and Antiandrogenic Progestins in Transgender Youth: Differential Effects on Body Composition and Bone Metabolism. Journal of Clinical Endocrinology and Metabolism 103(6): 2147-2156.

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I verify under the penalties for perjury that the foregoing representations are true.

Emma Hilton, PhD
27th June 2023

Appendix 1.
Emma Hilton Short-form academic CV

EMMA NIAMH HILTON
CURRICULUM VITAE

Faculty of Biology, Medicine and Health, School of Biological Sciences
University of Manchester, Manchester, M13 9PT
emma.hilton@manchester.ac.uk
http://www.manchester.ac.uk/research/emma.hilton

ROLES

04/2019 - present	Postdoctoral research associate, Division of Infection, Immunity & Respiratory Medicine, University of Manchester (funding from BBSRC; NC3Rs; Cystic Fibrosis Foundation).
01/2014 - 04/2019	Research Fellow, University of Manchester (funding from MRC, Newlife).
01/2010 - 01/2014	Stepping Stone Research Fellow, Genetic Medicine, University of Manchester (funded internally).
06/2003 - 12/2009	Postdoctoral research associate, Genetic Medicine, University of Manchester (MRC).

ACADEMIC QUALIFICATIONS

2004	Ph.D. Developmental Biology, University of Warwick, UK.
1999	B.Sc. (Honours) Biochemistry, University of Warwick, UK.

PUBLICATIONS (Google Scholar: Citations 1181, h-index 16)

Randles, M., Hamidi, H., Lausecker, F., Humphries, J.D., Byron, A., **Hilton, E.N.**, Clark, S.J., Miner, J.H., Zent, R., Humphries, M.J. and Lennon, R. Integrin-specific signalling pathways determine podocyte morphologies on basement membrane ligands. Submitted, Nat. Commun.

Hilton, E., Thompson, P., Wright, C. and Curtis, D. (2021). The Reality of Sex. Ir J Med Sci 190(4): 1647-1647.

Hilton, E. and Lundberg, T. (2021). Transgender women in the female category of sport: perspectives on testosterone suppression and performance advantage. Sports Med. 51 (2), 199-214.

Hindi, E., Williams, C., Zeef., L., Lopes, F., Newman, K., Davey, M., Hodson, N., **Hilton, E.**, Huang, J., Price, K., Roberts, N., Long, D., Woolf, A. and Gardiner, N. (2021). Experimental long-term diabetes mellitus alters the transcriptome and biomechanical properties of the rat urinary bladder. Sci Rep. 11(1):1-16.

Roberts, N.A., **Hilton, E.N.**, Lopes, F., Randles, M., Singh, S., Chopra, K., Coletta, R., Bajwa, Z., Hall, R., Yue, W. et al. (2019). Lrig2 and Hpse2, mutated in urofacial syndrome, pattern nerves in the urinary bladder. Kidney Int. 95(5):1138-1152.

Roberts, N.A., **Hilton, E.N.**, and Woolf, A.S. (2016). From gene discovery to new biological mechanisms: heparanases and congenital urinary bladder disease. Nephrol Dial Transplant. 31(4):534-540.

Stuart, H.M,, Roberts, N.A., **Hilton, E.N.**, McKenzie, E.A., Daly, S.B., Hadfield, K.D., Rahal, J.S., Gardiner, N.J., Tanley, S.W., Lewis, M.A. et al. (2015). Urinary tract effects of HPSE2 mutations. J Am Soc Nephrol. 26(4):797-804.

Roberts, N., Woolf, A. S., Stuart, H., Thuret, R., McKenzie, E., Newman, W. G., and **Hilton, E. N.** (2014). Heparanase 2, mutated in urofacial syndrome, mediates peripheral neural development in Xenopus. Hum Mol Genet 23:4302-4314.

Woolf, A.S., Stuart, H.M., Roberts, N.A., McKenzie, E.A., **Hilton, E.N.**, and Newman, W.G. (2013). Urofacial syndrome: a genetic and congenital disease of aberrant urinary bladder innervation. Pediatr Nephrol. 29(4):513-518.

Stuart H.M., Roberts, N.A., Burgu, B., Daly, S.B., Urquhart, J.E., Bhaskar, S., Dickerson, J.E., Mermerkaya, M., Silay, M.S., Lewis, M.A. et al. (2013). LRIG2 Mutations Cause Urofacial Syndrome. Am J Hum Genet. 92(2):259-264.

Maher, G.J., **Hilton, E.N.**, Urquhart, J.E., Davidson, A.E., Spencer, H.L., Black, G.C., Manson, F.D. (2011). The cataract-associated protein TMEM114, and TMEM235, are glycosylated transmembrane proteins that are distinct from claudin family members. FEBS Lett. 585(14):2187-2192.

Banka, S., Walter, J., Aziz, M., Urquhart, J. Vassallo, G., Clouthier, C.M., Rice, G., **Hilton, E.**, Will, A., Wevers, R.A. et al. (2011). Identification and characterisation of a novel inborn error of metabolism caused by dihydrofolate reductase deficiency. Am J Hum Genet. 88(2):216-225.

Briggs, T.A., Rice, G.I., Daly, S., Urquhart, J., Gornall, H., Bader-Meunier, B., Baskar, K., Baskar, S., Baudouin, V., Beresford, M.W. et al. (2011). Tartrate-resistant acid phosphatase deficiency causes a bone dysplasia with autoimmunity and a type I interferon expression signature. Nat Genet. 43(2):127-131.

Daly, S.B., Urquhart, J.E., **Hilton, E.**, McKenzie, E.A., Kammerer, R.A., Lewis, M., Kerr, B., Stuart, H., Donnai, D., Long, D.A. et al. (2010). Mutations in HPSE2 cause urofacial syndrome. Am J Hum Genet. 86(6):963-969.

Hilton, E.N., Johnston, J., Whalen, S., Okamoto, N., Hatsukawa, Y., Nishio, J., Kohara, H., Hirano, Y., Mizuno, S., Torii, C. et al. (2009). BCOR analysis in patients with OFCD and Lenz microphthalmia syndromes, mental retardation with ocular anomalies, and cardiac laterality defects. Eur J Hum Genet. 17(10):1325-1335.

Hanson, D., Murray, P.G., Sud, A., Temtamy, S.A., Aglan, M., Superti-Furga, A., Holder, S.E., Urquhart, J., **Hilton, E.**, Manson, F.D.C. et al. (2009). The primordial growth disorder 3-M syndrome connects ubiquitination to the cytoskeletal adaptor OBSL1. Am J Hum Genet. 84(6):801-806.

Tassabehji, M., Fang, Z., **Hilton, E.N.**, McGaughran, J., Zhao, Z., de Bock, C.E., Howard, E., Malass, M., Donnai, D., Diwan, A. et al. (2008). Mutations in GDF6/BMP13 are associated with vertebral segmentation defects in Klippel-Feil syndrome. Hum Mutat. 29(8):1017-1027.

Hilton E.N., Black, G.C., Manson, F.D., Schorderet, D.F., Munier, F.L. (2007). De novo mutation in the BIGH3/TGFB1 gene causing granular corneal dystrophy. Br J Ophthalmol. 91(8):1083-1084.

Hilton, E.N., Manson, F.D., Urquhart, J.E., Johnston, J.J., Slavotinek, A.M., Hedera, P., Stattin, E.L., Nordgren, A., Biesecker, L.G., Black, G.C. (2007). Left-sided embryonic expression of the BCL-6 corepressor, BCOR, is required for vertebrate laterality determination. Hum Mol Genet. 16(14):1773-1782.

Hilton, E.N., Rex, M., Old, R. (2003). VegT activation of the early zygotic gene Xnr5 requires lifting of Tcf-mediated repression in the Xenopus blastula. Mech Dev. 120(10):1127-1138.

Rex, M., **Hilton, E.N.**, Old, R. (2002). Multiple interactions between maternally-activated signalling pathways control Xenopus nodal-related genes. Int J Dev Biol. 46(2):217-226.

AUTHORED BOOK CHAPTERS

Hilton, E., Black, G.C.M., Bardwell, V. BCOR and oculofaciocardiodental syndrome. (2008/2013). Epstein's Inborn Errors of Development: The Molecular Basis of Clinical Disorders of Morphogenesis, 2nd/3rd edition, Oxford University Press, Oxford, UK.

GRANT INCOME

2016-2018	Newlife (£115,735). Towards novel therapies for an inherited congenital neuropathy affecting the urinary bladder. Woolf, Newman, Kimber, **Hilton** (Co-app).
2014-2016	MRC (£507,695). Molecular bases of congenital bladder diseases. Woolf, Newman, Gardiner, **Hilton** (Research Co-I).
2010-2013	KRUK (£180,000). Urofacial syndrome (UFS): a novel genetic model to understand human renal tract function and malformation. Newman, Woolf, McKenzie, **Hilton** (Co-app).
2010-2014	University of Manchester (£salary + £40,000 project costs). Xenopus as a model organism for human development and disease. **Hilton** (Stepping Stone Fellowship Award).
2008-2010	Newlife (£100,000). The role of BCL-6 corepressor-modulated TGFβ signalling in MCOPS2 and other microphthalmia syndromes. Black, Manson, **Hilton** (Co-applicant).

Appendix 2.
Hilton and Wright, 2023. “Two sexes”. Sex and Gender: A Contemporary Reader. Routledge, Oxford, UK.

TWO SEXES
Emma Hilton and Colin Wright

‘Why the sexes are, in fact, always two.’
Sir Ronald Fisher, 1930

Sex is an evolved system function common to almost all complex life on earth, a fact that is often forgotten by postmodernist commentators intent on framing sex as a human-centred, human-invented—and thus malleable—construction of scientific understanding. The aim of this essay is to review the biological understandings of the phenomenon that is sex.

In the first section, we will answer the question: why does sex exist? We will explain its evolutionary origins, and the binary gamete system on which ‘female’ and ‘male’ are founded. To finish this section, we explore some of the diversity of sex—female and male—in the natural world, to understand how reproductive bodies in almost all complex life are organised around these functional roles. In the second section, we will focus on developmental biology and how sex manifests in humans: how we make babies and how female and male humans develop. In the final section of this chapter, we will critique emerging ideological misinformation about sex, particularly in humans, addressing arguments that, for example, assert sex as a social construct or seek to deconstruct standard understandings of sex as a binary phenomenon. We will highlight fundamental misinterpretations of sex and its associated characteristics, the unscientific focus on those people with atypical sex development and the dangers of viewing sex as a statistical outcome.

The incursion of ideological misinformation about sex into the academic fields of medicine and biology generates confusion in research and presents potential for harm. ‘Sex matters’ in basic and applied health research (Wizemann and Pardue, 2001) and the US National Institutes of Health, the EU Commission, research funding bodies and academic journals increasingly demand that researchers account for ‘sex as a biological variable’ in their research design, analyses and reporting, whether they include studies of whole organisms or cell lines. However, progress is slow. The UK NHS maintains a confusing system where biological sex cannot be disaggregated (Forstater, 2021), and the World Health Organisation promises to, ‘achieve greater impact on health [using] sex disaggregated data’ (WHO/Health topics/Gender) while simultaneously updating guidance to assert that, ‘sex is not limited to male or female.’ (WHO, 2022). We have publicly argued that, from the wider scientific perspective, ideologically-driven scientists are in danger of sacrificing, ‘empirical fact in the name of social accommodation’ and this is both, ‘an egregious betrayal to the scientific community they represent’ and, ‘undermines public trust in science.’ (Hilton and Wright, 2020). By re-asserting biological knowledge established over the preceding centuries and countering deconstructive discourse, this essay may be considered a reconstruction of sex.

A note on language. Physiologist Ernst Wilhelm von Brucke noted that, ‘Teleology is a lady without whom no biologist can live. Yet he is ashamed to show himself with her in public.’ (Davis and Uhrin, 1991). It is possible in discussions of evolutionary biology to avoid teleological language, but sentence constructions are often overly verbose and clunky. For ease of readability, we sometimes use language that is teleological in tone, but, in the words of zoologist

Simon Maddrell (1998), ‘This should not be taken to imply that evolution proceeds by anything other than from mutations arising by chance, with those that impart an advantage being retained by natural selection.’

WHAT IS SEX?

And why does sex exist? Remarkably, it is not uncommon to find purportedly-scientific articles about sex that neglect to mention its evolved function of reproduction (for example, Ainsworth, 2015; Sun, 2019). Indeed, that science communicators writing about sex often focus only on lists of physical characteristics associated with sex means that, despite the author claims, such articles are not actually addressing the biological phenomenon of sex—what is it? why does it exist? why do humans have sexed bodies? Rather, they are addressing how the sex of a given individual may be identified via some checklist of physical features that—ironically—could only have been created by understanding how those physical features are associated with reproductive function. We return to this conflation of sex (what it is) with the physical characteristics associated with sex (how we recognise the sex of a given individual) in the final section of this essay.

Reproduction The phenomenon of sex is rooted in reproduction, the process by which new individuals are produced from a parent or parents. There are two types of reproduction in the natural world: asexual and sexual. In asexual reproduction, a parent replicates all of its genetic information and generates new individuals by processes such as binary fission—the division of a parent cell into two identical (or, at least, very similar) cells, observed in bacteria—and budding—which produces a new individual from a parental outgrowth, observed in yeast.

In asexual reproduction, offspring receive a full set of genetic information from a single parent; it follows that offspring are genetically-identical clones of that parent. Individual expansion, via asexual reproduction, of a genetically-identical (or genetically-similar) population is a relatively low-cost biological burden, and rapid to enact; consider how quickly mould, which can reproduce asexually via the production of independent spores that populate the local environment, can colonise a loaf of bread, or how quickly bindweed can aggressively invade a garden by sending out roots from which new individuals grow. There are also parental benefits, as each parent passes on all of its genetic information to the next generation.

Yet despite the existence of a low-cost and rather straightforward method of reproduction, the natural world is dominated by species that employ a different reproductive strategy: sexual reproduction.

Unlike asexual reproduction, sexual reproduction involves two parents, almost always from two different classes of individuals called ‘females’ and ‘males’; each contributes half of their genetic material—carried on chromosomes—resulting in the generation of a new and genetically-unique individual. The mixing of genetic material from each parent (and thus, the beginning of a new individual) is achieved, in a process called ‘fertilisation’, by the fusion of two specialised cells called ‘gametes’. Gametes are a unique cell type within sexually-reproducing species and the function of the gamete within any individual is singular—to effect sexual reproduction.

Sexual reproduction is biologically-costly to individuals, not least because mating requires resources (for example, energy expended on locating a mate) and carries health risks (for example, disease transmission and exposure to predators). In most sexually-reproducing populations, half of the offspring will be males who cannot themselves bear offspring; thus, these populations experience lower growth rates than found in asexual populations, where all offspring

can themselves bear offspring (‘the cost of males’; Maynard Smith, 1978). Furthermore, genetic relatedness between parent and offspring is much lower than in asexually-generated clones, and each individual must therefore invest biological resources in producing at least two offspring to have any chance of passing all genetic material to the next generation. Explaining these costs—the ‘queen of problems in evolutionary biology’ (Bell, 1982)—has challenged evolutionary biologists; given the disadvantages, why did sexual reproduction evolve from asexual reproduction to become, by far, the most common method of reproduction in complex species?

The fusion of two gametes means that the new individual possesses a chromosomal makeup different to either parent and, given recombination between the chromosomes in each parent, chromosomes that carry different combinations of genetic material to either parent. The prevalence of sexual reproduction indicates a strong evolutionary advantage for this mechanism of reproduction that mixes genetic material. Such advantage is typically conceptualised as novel combinations of genes and changes in them (mutations) upon which evolutionary selection can act, the foundation of Darwin’s theory of evolution by natural selection (Darwin, 1859), and divided into two broad hypothesis domains: the accumulation of beneficial genetic changes and the removal of detrimental genetic mutations. Accumulation of beneficial genetic traits are advantageous in adaptation to changing environments (the ‘Fisher-Muller model’; Fisher, 1930; Muller, 1932) or co-adaptation, in an arms race, alongside interacting species who are trying to harm you (van Valen, 1973; delightfully called the ‘Red Queen hypothesis’ after Lewis Carroll's character in Through The Looking Glass, who observed, ‘Now, here, you see, it takes all the running you can do, to keep in the same place.’) However, the benefits of bringing together useful genetic traits during sexual reproduction must be balanced by the possibility that already coexisting beneficial traits are separated among offspring (Desai and Fisher, 2007). By contrast, harmful genetic mutations—those that compromise evolutionary fitness—must be weeded out to prevent them from accumulating in a population (see ‘Muller’s ratchet’, from Muller, 1964; also ‘Kondrashov's hatchet’, after Kondrashov, 1988).

The fitness advantages conferred by sexual reproduction explain its near-ubiquity among complex species. Indeed, even plants and simple animal species that typically reproduce asexually in stress-free environmental conditions to which they are comfortably adapted can switch to sexual reproduction during times of stress or environmental change, when genetic mixing may produce a survival advantage among offspring (for example, Becks and Agrawal, 2010). So successful an evolutionary strategy is sexual reproduction that many complex species, including humans, have completely lost the ability to reproduce asexually. No wonder Erasmus Darwin remarked that, ‘Sexual reproduction is the chef d'oeuvre, the masterpiece of nature.’ (Darwin, 1800).

Gametes and sexes While genetic exchange mechanisms exist, well-studied in bacteria and virus-host interactions, where DNA is transferred between different individuals in a non-sexual fashion (Callier, 2019), the evolutionary root of sexual reproduction via specialised gametes lies with the evolution of multicellularity, at least 1.5 billion years ago (Fu et al., 2019). In simple species like yeast (who can reproduce both sexually and asexually), all gametes are structurally similar; this is called ‘isogamy’. However, successful gamete pairing and fusion may be limited by molecular compatibility—mediated by various proteins on the cell surface (for example, Lipke and Kurjan, 1992)—between the cells of the parents. Such compatibility groups are described as ‘mating types’, usually labelled by a system of numbers (for a primer on mating types, see Fraser and Heitman, 2003). The number of mating types within a species can be thousands, and they functionally promote genetic diversity within a population by preventing gamete fusion

between genetically-similar parents. Isogamy is thought to be the ancestral state for sexual reproduction and remains common in simple sexually-reproducing species like yeast.

For an excellent overview of gamete evolution, see Lehtonen and Parker (2014) and references therein. Briefly, modelling of evolutionary scenarios for a variety of gamete characteristics shows that a binary system of gametes is optimal; that is, large gametes and small gametes, with gamete fusion occurring only between one small and one large gamete (not small-small or large-large fusions). We call this binary system of gamete fusion ‘anisogamy’.

In species with two gamete types, the large gamete (and associated biology) is termed ‘female’ and the small gamete (and associated biology) is termed ‘male’. In animals, the female and male gametes take the familiar forms of egg and sperm, respectively. In plants, the female and male gametes are contained in the ovules and pollen, respectively. That two different gametes form the optimal arrangement for sexual reproduction is understood in terms of gamete specialisation. The female gamete, with greater physical volume, single-handedly provides to the developing embryo basic cellular components, many molecules and signals required to direct early growth and energy-creating units called ‘mitochondria’. Strict uniparental—specifically, maternal—inheritance of cellular components—commonplace in anisogamy—is presumed favourable for embryo health by eliminating any biological compatibility between mitochondria (Greiner et al., 2015) and eliminates wasting when both parents invest resources in these components. In contrast, the male gamete sacrifices contribution to offspring beyond the chromosomes contained in its nucleus. Male gametes in many species have typically become specialised for mobility to better access female gametes—consider the tail-like structures of sperm that propel it towards the egg (Lessels et al., 2009) and pollen grains sticking to bee legs (Hu et al., 2008)—and created in large numbers to improve the chances of both an encounter with a female gamete and the outnumbering of small gametes from other males (Parker and Lehtonen, 2014).

Anisogamy is the evolutionary origin of the two sexes—the reproductive roles associated with female or male gamete contributions to offspring. The evolution of gametes into two non-overlapping, morphologically-distinct types necessitates specific cellular and tissue systems to produce either one or the other gamete and favours the subsequent evolution of anatomy that facilitates successful fertilisation events. The evolution of separate sexes is thought to have arisen multiple times in plants and animals, suggesting an evolutionary benefit. Common explanations include higher individual fitness when an individual is specialised for a single reproductive role, rather than trying to balance resources between both male and female functions (Charnov, 1982). In fact, given differential gamete morphology, the subsequent divergence into two separate sexes of individuals has been described as, ‘an almost inevitable consequence of sexual reproduction in complex multicellular organisms.’ (Lehtonen and Parker, 2014). Extending from mere inevitability to essentiality of outcome, Kashimada and Koopman (2010) state that, ‘the development of two sexes is observed in most animals and is essential for their survival and evolution.’

Why this almost inevitable divergence into just two sexes of individuals has occurred—repeatedly in evolutionary history—is the subject of much research. To answer this question, we must review the established knowledge on gamete evolution—the halving of genetic material, uniparental inheritance of intracellular components—and interrogate under what conditions could a third reproductive role—a third sex—evolve and what function could it have? Indeed, an exploration of this question was the prompt for the opening chapter quote. That is, ‘No practical biologist interested in sexual reproduction would be led to work out the detailed consequences experienced by organisms having three or more sexes; yet what else should he do if he wishes

to understand why the sexes are, in fact, always two?' (Fisher, 1930). And from science to science fiction, this question is, wonderfully, puzzled over by Kurt Vonnegut's Billy Pilgrim who, when considering the Tralfamadorians in Slaughterhouse-Five, surmised, 'They said their flying-saucer crews had identified no fewer than seven sexes on Earth, each essential to reproduction. Again: Billy couldn't possibly imagine what five of those seven sexes had to do with the making of a baby, since they were sexually active only in the fourth dimension. [...] It was gibberish to Billy.'

Sexual systems and bodies Across almost all complex life, there are precisely two types of gamete—and thus two and precisely two sexes. But this does not impose restrictions on how sex is allocated in different species. Evolutionary biologist Lukas Scharer illuminates, 'The male and female sexes are not two types of individuals; they actually represent two different reproductive strategies, and in many organisms, these two strategies are distributed among individuals in a population in a variety of ways.' (Scharer, 2017). That is, across the natural world, there is great diversity (and ingenuity, if one can—teleologically, of course—describe characteristics favoured by natural selection as 'ingenious') regarding the allocation of male and female sexes within and between individuals and across populations.

A 'sexual system' describes the physical and functional interactions of the two sexes at the individual and population level. We have learned that the evolution of separate sexes of individuals—a state called 'gonochorism'—is near-ubiquitous in animals; individuals within a gonochoristic species comprise two anatomic classes divided by reproductive role. Typically, male or female sex is fixed early in embryonic development and immutable to change during the lifespan of any individual, even though, of course, the physical characteristics associated with sex may be subject to expected age-related changes or changes acquired via injury or disease (or, at the hands of humans, surgery).

Humans cannot be hermaphrodites—individuals who fulfil both male and female reproductive roles in their lifespan—though hermaphroditism is a natural body plan in many anisogamous species. Many plants—particularly flowering plants—and (few) less complex animals exist as simultaneous hermaphrodites, with both female and male sexes manifested in the same flowers and/or same individual plant or animal at the same time of life. Many aquatic species—most notoriously, clownfish—are sequential hermaphrodites, where changes in reproductive role during the lifespan ('sex change') are evidenced by the switch from male to female (in the case of clownfish) or female to male gamete production, underpinned by anatomical changes in gamete-producing tissues (gonads). In the case of clownfish, this switch of sex (male to female) is driven by the loss of the single breeding female from the colony (Casas et al., 2016). Sequential hermaphroditism appears most common in species where males and females have the same excretory structures for eggs and sperm, and 'sex change' requires no or minimal remodelling of gross anatomy. For example, clownfish fertilisation is external, and male and female clownfish both have a similar ductal system that allows the sperm and eggs, respectively, into the aquatic environment. With highly-specialised and qualitatively-different reproductive anatomies, neither obviously nor easily remodelled post-development, 'sex change' in humans is impossible.

Evolution provides a dazzling array of anatomies and appearances. It is often true that gonochoristic males whose reproductive role is to contribute sperm have evolved appendages for direct introduction of that sperm into females, while the females of many species have evolved internal biology that receives sperm and, in the case of viviparous mammals who give birth to live young, protects the developing offspring from the outside world. But appearances can be

deceptive. For example, male seahorses have a brood pouch in which developing baby seahorses are incubated, a functional role more usually associated in the natural world with female individuals. However, these seahorses are the sex class that contribute sperm to the offspring, and it is that, not their gross anatomy, which defines those individuals as male. Another curious example is that of female spotted hyenas, who have a hyper-enlarged clitoris that resembles a penis, yet they produce eggs that are fertilised by a male hyena and are, by definition, female. Human-centred biological expectations about anatomy, which include, for example, pregnancy in females and penile appendages in males are undoubtedly too narrow to capture the diversity of sexed bodies in the natural world.

Hermaphrodites incorporate both male and female sexes, and gonochorists one or the other. And while gonochorism and simultaneous hermaphroditism represent stable arrangements of the two sexes within a species, there are many that buck these trends in their individual composition. For example, there are species composed of females and hermaphrodites (McCauley and Bailey, 2009), of males and hermaphrodites (Weeks et al., 2009), and of males, females and hermaphrodites (Oyarzun et al., 2020). That is, the two sexes can be differentially-allocated in individuals and between species. Yet, despite the variety of bodies and sexual systems found in the natural world, their organisation around two and only two sexes is a fundamental feature. Reproduction within and between individuals occurs by the meeting of female and male gametes, one of each type, in that precise combination, in a pattern recapitulated across almost all complex life. The binary system of sex is an evolutionary thread stitched through life on earth.

HUMAN SEX

We have established what sex is, that sex describes reproductive role by reference to gamete type, and that there are—and can only be—two sexes. We have also described some of the fascinating manifestations of the two sexes within individuals and within populations. In this section, we turn to developmental biology—the study of how organisms grow and, increasingly, how they age—which is replete with examples of complexity of form built from simple biological principles. The development of the reproductive human is one such instance.

The developmental biology underpinning this section is largely sourced from standard reference textbooks in the field. Readers may also wish to explore Baresi and Gilbert’s Developmental Biology (online at the National Centre for Biotechnology Information) and Wolpert’s Principles of Development.

Making a baby Humans are mammals and are—like almost all animals—divided into two classes of individuals according to reproductive role. In humans, the act of reproduction itself requires, in the first instance, male sperm to fertilise female eggs, achieved during intercourse between two sexually mature people. Male reproductive anatomy includes testes, contained in a sac of skin called the ‘scrotum’, that make sperm, delivered to the outside world through the penis. Both testes and penis are external organs, housed outside the male body, while female reproductive anatomy is almost wholly internal. It comprises ovaries that periodically release mature eggs, collected by the nearby oviducts (also called Fallopian tubes) and transported towards the uterus, the hollow muscular organ in which, in the event of a successful fertilisation event, a baby will grow. The uterus connects, via the cervix, to the vagina, which exits the body at the vulva, incorporating the clitoris and the urethral opening, surrounded by folds of skin called labia.

During intercourse, male sperm is mixed with water and lubricants from the seminal vesicles and prostate gland (to create semen), and the penis delivers the resulting semen into the female body via ejaculation. Semen moves through the cervix and uterus to the oviducts, where, should a mature egg be ready, fertilisation occurs. The fertilised egg is transported then implanted into the uterine wall, and commences development proper - at this stage, the female is pregnant. In the absence of a successful fertilisation event, the female body, having already prepared a blood-rich, spongey uterus lining suitable for implantation, breaks down this lining and expels it via the vagina during menstruation. In humans, gestation—the growing of a baby within the pregnant female uterus—lasts around nine months, after which the female gives birth, typically via the vagina (although surgical interventions like caesarean section, where the baby is removed via an incision through the uterus wall, may be necessary in negative medical circumstances or elected as a preference).

Sex determination Reproductive anatomy develops in utero, in a series of complicated yet elegant anatomical steps. The first step in reproductive development, however, is the determination of the future sex of a new embryo: female or male? In humans, sex is genetically-determined at fertilisation via the XY determination system of sex chromosomes. Females possess two X chromosomes, while males possess one X and one Y, with the Y chromosome carrying male-specific genes that activate male development. Given that sex chromosomes, like all other pairs of chromosomes, are divided individually when gametes are made, each human egg contains one X chromosome (and females are called ‘homogametic’) while human sperm contains either an X or Y chromosome (with males termed ‘heterogametic’). Offspring sex is thus dependent on whether an egg receives, at the moment of fertilisation, either an X or Y chromosome from the sperm.

The pattern of chromosomes within an individual is called a ‘karyotype’. Like all chromosomes, sex chromosomes carry genes. In humans, a key sex-determining gene is called SRY (sex-determining region Y) and it is, in genetically-healthy individuals, carried by the Y chromosome (Kashida and Koopman, 2010; Sinclair et al., 1990). The protein product of the SRY gene acts as a ‘master switch’ for male development, initiating a cascade of molecular genetic signals that drives the first anatomical step towards a sexed human body, gonad differentiation.

Embryonic development Gonad differentiation occurs at around six weeks in utero, when a bipotential pair of gonads—small buds of tissue in the abdominal cavity—are triggered to differentiate into ovaries or testes, the gamete-producing tissues in females and males, respectively. XY embryos carrying a functional SRY gene will trigger differentiation of testes via a network of molecular signals; in the absence of SRY activity, XX embryonic gonads begin to differentiate into ovaries, activating distinct molecular signals for that developmental pathway (Lecluze et al., 2020; Mamsen et al., 2017). There is feedback between these differentiation pathways; for example, a signal required for ovarian development—and the later maturation of eggs—also suppresses early testes differentiation (Jaaskelainen et al., 2010).

In embryological terms, gametes do not originate in the growing gonads. Rather, specialised stem cells migrate into the differentiating gonad region where they are embedded as the precursor cells that will ultimately become eggs or sperm, depending on gonad type (Magnusdottir and Surani, 2014). Ongoing gonad development into mature egg- or sperm-producing tissues relies on the differentiation of sex-specific gonad cell types, a process requiring tissue-specific hormone action. However, gonad differentiation into ovaries or testes also directs, via that sex-specific hormone milieu each generates, downstream events in reproductive anatomy development coordinated with future gamete type. That is, ovaries fated

to produce eggs will direct ongoing female development and testes fated to produce sperm will direct ongoing male development. In this sense, gonads can be considered as organiser tissues, coordinating the development of a reproductive system that integrates future gamete type with relevant reproductive anatomy; the absence of future gamete function—infertility—is no barrier to understanding the sex of human.

The first embryonic targets of gonadal organisation, from around eight weeks in utero, are two pairs of ducts running alongside the gonads called the paramesonephric and mesonephric ducts, which will grow into female or male internal genitalia, respectively. Both female and male embryos develop both pairs of ducts in early development; after gonadal differentiation, sex-specific hormonal action promotes growth of one pair over the other. Male testes secrete two major hormones that act upon these pairs of ducts. Testosterone promotes the growth of the mesonephric duct into male internal genitalia, and secreted anti-Mullerian hormone triggers degeneration of the paramesonephric duct, thus eliminating the duct that would develop into female internal genitalia. In females, the presence of ovaries means there is little testosterone to promote growth of mesonephric duct structures, nor anti-Mullerian hormone to trigger degeneration of paramesonephric duct structures. The female hormone environment thus permits growth of female internal genitalia, while the mesonephric duct (and the potential for male internal genitalia) degenerates.

The second embryonic target of gonadal organisation, from around 10 weeks in utero, is the development of external genitalia. The external genitals—vagina, clitoris and labia in females and prostate, penis and scrotum in males—derive from shared precursor tissues called the genital tubercle and urogenital fold. Under the influence of sex-specific gonadal hormones, these tissues are moulded into male or female form. Specifically, a derivative of testosterone (dihydrotestosterone) is produced locally—from testosterone—in the precursor tissues in males, and this derivative is a potent inducer of male external genitalia. In the converse situation, low testosterone and low dihydrotestosterone in females permits this precursor tissue to develop into female external genitalia. Given that male and female external genitalia develop from the same embryonic tissue under differential hormonal influences, analogous structures can be identified: the clitoris and penis share many structural features, while the labia represents an unfused version of the scrotum.

The sex of a newborn baby is routinely and reliably observed at birth by visual and palpable (‘touch’) assessment of external genitalia. Increasingly, the sex of a baby is identified in utero by observation of external genitalia or detection of sex chromosome karyotype/SRY gene presence. This is a matter of observation, woefully mischaracterised by the term ‘assignment’. The language of ‘assignment’ has been co-opted from serious medical decision-making in the case of clinical pathologies of the reproductive system (discussed below).

Puberty and secondary sex characteristics The development of reproductive anatomy in utero is called ‘primary sex development’, and the outcome is a body that has the potential to fulfil the male or female reproductive role. Human sex development undergoes a second phase of development at puberty, between the ages of 10-18 years old. This phase of secondary sex development generates divergence between the body shapes of females and males—a phenomenon called ‘sex dimorphism’—that has evolved under selection pressure to increase one’s likelihood of mating, following two broad strategies: be the most attractive or the most dominant. Both females and males gain height and bone density, experience the onset of libido, and experience typical teenage symptoms like acne and body odour. Under the influence of sex-specific gonadal hormones, female reproductive anatomy matures, ovulation and menstruation

commence, hip width increases, and breast tissue develops, in preparation for future motherhood. As well as experiencing male-typical maturation of reproductive anatomy (increase in testes volume and penile length), males gain greater height than females, grow facial hair, develop deeper voices, broader shoulders, and acquire far larger amounts of skeletal muscle than females.

Atypical sex development We have described the typical reproductive anatomy and sequence of events during development in healthy human beings. However, as a system with multiple biological inputs, steps and components, atypical or pathogenic development of reproductive anatomy can occur; in short, there are many points at which reproductive development can go awry. Collectively, medical conditions resulting from atypical reproductive development are called disorders (or, in patient-facing language, differences) of sex development (DSDs). There are around 40 known DSDs occurring in humans, most a result of mutations in genes required for the healthy development of reproductive anatomy in utero (Arboleda et al., 2014). The category of DSDs is broad, and it spans simple anatomic and hormone differences in otherwise healthy individuals to disorders with acute clinical sequelae that can cause postnatal harm or even death, and that need ongoing management throughout life.

Historically, DSDs have been described by terms such as ‘hermaphroditism’ and—currently falling into disuse—'intersex’. These terms are now deemed clinically-inaccurate and stigmatising to patients. Current nomenclature to categorise DSDs references karyotype and gonad status. Thus, the overarching categories are sex chromosome DSDs, XY DSDs and XX DSDs. For example, sex chromosome DSDs are exemplified by Turner syndrome and Klinefelter syndrome, where patients have irregular numbers of sex chromosomes and develop along typical female and male developmental trajectories, respectively, but experience hormonal issues that compromise sexual maturation and fertility. Other DSDs include conditions where female embryos are exposed to excessive testosterone in utero and develop an enlarged clitoris (an XX DSD called congenital adrenal hyperplasia) or where male embryos fail to produce the dihydrotestosterone required for penis growth (an XY DSD called 5 alpha reductase deficiency). Excellent resources on DSDs and their developmental etiology have been compiled, in collaboration with expert clinicians, by the UK charity DSD Families, and are available at their website.

The frequency of DSDs in the general population is the subject of much misinformation. Fausto-Sterling and her associates have defined as ‘intersexual’ any deviation from ‘ideal, Platonic’ male and female bodies, and arrived at a frequency of 1.7% of the population (Blackless et al., 2000; Fausto-Sterling 2000). Such a loose definition of DSDs captures a large number of people with no biologically-meaningful ambiguity of sex in any aspect of their development (most egregiously, the vast majority of this reported frequency are unambiguous females, often mothers, who have late-onset adrenal hyperplasia and, at some point post-birth, experience elevated testosterone levels as a result of an adrenal problem). This frequency of 1.7% was revised by Hull and Fausto-Sterling (2003) who, after identifying numerous flaws in the original studies, like failing to account for the sex-specific nature of many DSDs, revised the frequency to 0.4% of the population.

When assessing DSD frequency rationally restricted only to those individuals with ambiguous sexual anatomy or who exhibit a disparity between their reproductive (gondal) sex and external genitalia, the original frequency of 1.7% drops dramatically down to approximately 0.018 percent (Sax, 2002). That is, despite atypical sex development, almost all people are identifiable as either female or male. Within modern medicine, workflows to identify internal genitalia, karyotype and

hormonal profiles exist to identify sex in ambiguously-presenting people, and understanding DSDs within the framework of typical developmental trajectories of females and males aids not only diagnosis of these clinical disorders but also informs prognostic decisions regarding management of specific conditions in terms of sexual function and fertility prospects. Nonetheless, the inflated frequency of 1.7% is routinely-cited as definitive (for example, by Amnesty in 2018).

SEX MYTHS

In 2021, in a letter published in the Irish Journal of Medical Science (the official organ of the Royal Academy of Medicine in Ireland), we argued that, 'Public discourse around sex increasingly seeks to deny basic facts of human biology.' (Hilton and Wright et al, 2021). The driver of this anti-science movement is gender identity ideology, which claims that a privately held identity regarding one's sex is the ultimate definer of one's sex. That is, if a person identifies (in some internal, unverifiable sense) as female or male, that person literally is female or male. The overarching aim of gender identity ideologists is to deny that sex—reproductive role and associated characteristics—exists as a natural biological category. The intent behind such a belief appears to be to undermine the common scientific understanding and validity of viewing females and males as discrete biological categories in favour of a wholly subjective and unfalsifiable categorisation scheme based on one's personal and internal sense of self—gender identity. In this section, we will critique emerging misunderstanding, real or contrived, around sex.

Myth: sex is a composite score of body parts Underpinning ideological misrepresentations about sex is the conflation of sex (what is female?) with the physical characteristics associated with sex (how do we recognise female people?). That is, sex is not presented as anatomical patterns that develop in a co-ordinated fashion within the framework of an evolved function but as a checklist of seemingly-independent physical characteristics. This is often explicit; a Nature (2018) editorial asserted sex is, 'a classification based on internal and external bodily characteristics.' in a piece that failed to mention reproduction, the function of sex, or why humans have sexed bodies. And failed to acknowledge the obvious follow-up question: a classification based on internal and external bodily characteristics in which species? Of course, the reference species is assumed human, a peculiarly self-centred view of a biological phenomenon common to almost all complex life. In this sense, the conflation of sex with characteristics associated with sex retrospectively requires the redefinition of sex in every species on earth deploying anisogamy as a means of reproduction, while ignoring the unifying features shared by all.

Writing for The Skeptic in 2021, Hearne accurately defines 'female' as, 'organisms whose gametes are [...] ova or eggs.' yet immediately follows with, 'Unless you are a fertility doctor, it's unlikely you will encounter too many ova, so we must be using other definitions in everyday life.' While it is true that gamete type is not directly assessed in strangers, it does not follow that we use alternative 'definitions' when identifying the sex of a person; more accurately, we use alternative sex characteristics, those that arise from the organisational effects of the gonads (which also dictate gamete type) during primary and secondary development. Hearne claims that features like external genitalia—routinely covered—and breast size—plumped by bras—are insufficient to identify the sex of a stranger, and that we do so by features such as, 'amount and distribution of muscle and fat, the length and distribution of hair, the height and so on.' This is true; in fact, psychiatrist Nirao Shah, who studies behavioral differences between males and females, considers, 'correctly identifying [...] sex [is] a fundamental decision animals make.' (Goldman, 2019). Alongside basic assessments of body shape like shoulder and hip width, humans are expert with faces; sex identification is, 'an automatic and effortless aspect of face

perception' triggering differential brain activity (Kaul et al., 2011). Intriguingly, females are consistently better than males at recognising female faces, even in the absence of (often) gendered cues like hair length (for example, Lewin and Herlitz, 2002). Humans also assess movement like walking gait in sex identification (Pollick et al., 2005). However, none of these data points is, as Hearne's logic would have it, a 'definition' of sex, in the same way that observing the texture and density of a rock allows us to identify it as igneous, where 'igneous' is defined as a rock generated from volcanic lava.

Changing the definition of sex from function to form—explicit in pieces with titles like 'Sex Redefined' (Ainsworth, 2015) and where function is often discarded as irrelevant—is a necessary foundation upon which the deconstruction of sex as a biological category is built. Following the redefinition of sex as a checklist of physical characteristics, claims regarding variability of characteristics can flourish, along two lines of argument. First in line are those people with DSDs who have atypical reproductive development. The description of sex characteristics in people with DSDs sometimes disaggregates a reproductive system into constituent parts like 'genetic sex', 'gonad sex', and so on, to better understand incongruent features, clinical management and prognostic outcomes in people with DSDs (for example, Arboleda et al., 2016). For nearly all people, these constituent parts are aligned—or at least not divergent in any meaningful way—and disaggregation has no utility. If such disaggregation can be considered useful, it is not in the redefinition of female and male sexes, but in the refinement of workflows that generate a complete clinical picture for those people with DSDs. However, since the coining of 'gender identity' by John Money in the 1960s, component parts of sex have occasionally included concepts of 'psychological' and 'social' sex (Moore, 1968), paving the way for 'identity' to be considered a sexed characteristic.

The second line of argument evokes those sex characteristics, like height and hormone levels, that can overlap between the sexes, to attempt to demonstrate that there is no clear boundary between the female and male sexes in humans, and that, 'there is no one parameter that makes a person biologically male or female.' (Elsesser, 2020). The aim here is to destabilise the established categories of female and male. It is, of course, true that, for example, many females are taller than many males, or that some males have low levels of testosterone more typical of the female sex. However, such arguments fail to acknowledge an elephant in the room—we can only know that males are typically taller and have higher testosterone levels than females if we have a means to divide and measure humans by sex, independent of height and testosterone level. And it is centuries of knowledge accrued by the study of sex as a functional property of a species, and the anatomic/molecular organisation of the human species around that evolved function, that serves as that reference point. Put simply, it would be impossible to claim that low and high testosterone levels are correlated with being female and male, respectively, unless the categories female and male already had established meanings that testosterone levels were being correlated with. And the same holds for every other sex correlate.

Myth: sex is not binary Having remapped the definition of sex from function to form, introduced exceptions—arising from clinical disorders—to Fausto-Sterling's 'Platonic ideal', and attempted to blur category boundaries in healthy humans with trivial observations of naturally-overlapping sex characteristics, various commentators have attacked the phenomenon of sex as a binary system, often failing—deliberately or otherwise—to understand what the term 'binary' means when applied to sex. Writing for the Guardian in 2015, Heggie claims 'binary sex' means, 'the idea that there are men and women and they can be clearly distinguished.' (Heggie, 2015). Cade Hildreth (2022) claims that, 'sex is not binary because people cannot be grouped into two separate, non-overlapping groups.' These are straw man arguments.

The functional system of sex is routinely-described as ‘binary’ (including, on many occasions, by us). The use of ‘binary’—meaning, ‘of, pertaining to, characterised by, or compounded of, two’ (Oxford English Dictionary)—in this context intends to indicate, simply, a biological system with two components, and follows the same etymological pattern by which, for example, a system composed of two stellar masses is described as a binary star. Use of the word ‘binary’ operates at a system level across all species employing anisogamy.

However, having constructed a straw man argument that sex in humans is not binary, rejection of the term ‘binary’ is extended into rejection of ‘two’ itself, and the substitution of ideological framings of sex that move the conversation far from biological reality. Many interlocutors posit quantitative descriptions of sex as the necessary alternative to categorical descriptions. The most common quantitative (continuous) data distribution used to frame sex is a bimodal distribution, whereby various quantifiable traits associated with sex, such as adult height and testosterone levels, are conceptualised as multiple, overlapping distributions. These overlapping distributions of individual traits are purported to generate two modes that represent the average or typical female and male (as described by a combination of their average or typical sex characteristics), while shoulders for each mode permit for variation of sex characteristics. Routinely plotted on a horizontal axis crudely labelled ‘sex’, this framework gives rise to the premise that one’s sex is a statistical score generated by measuring multiple quantifiable characteristics. For a widely-circulated conceptualisation of ‘bimodal sex’, Hildreth (2022) describes the modes as, ‘peaks in a graph [that] represent probability clusters.’ Further to claims that sex is bimodal are claims that, ‘The science is clear—sex is a spectrum.’ (Brusman, 2019), an expression of a continuous distribution that replaces modes with, in the words of Brusman, ‘unlimited options.’ The corollary is that the sex of every human is unique to that individual, or, in the words of Fausto-Sterling when considering The Five Sexes, Revisited (2000), ‘Rather than identify a specific number of sexes [...] sex and gender are best conceptualized as points in a multidimensional space.’

The outcome of categorising sex as the sum of continuous descriptions of sex traits is that every person is scored as some percent male or female. The often-denied logical progression of such scoring is that a male with lower than average testosterone, petite stature, or a smaller than average penis, is shifted away from the male mode towards the female mode (typically occupied by people with low testosterone, petite stature and no penis). Such males, by this framework, are scored as ‘more female’ than counterparts with average or high testosterone, great stature and large penises. These damaging judgments equally extend to females with enlarged clitorises, small breasts or increased musculature, who, by the above logic, are scored as ‘more male’ than their larger-breasted and less athletic counterparts.

As sex within a continuous framework becomes a matter of sliding people left or right towards and from typical female and male, the middle of this distribution is cast as the no man’s land where—plus ça change—people with DSDs are placed. For those with little comprehension of DSDs beyond vague imaginings that people with DSDs have ‘both sets’ of genitals, this is intuitive. However, DSDs do not present as random combinations of primary and secondary sex organs, and neither do they simply differ by degree from one another. Rather, DSDs represent dozens of conditions with unique etiologies that manifest in disparate ways. There is no single medical category that is ‘intersex’ nor is there a robust method of ordering them, as would be necessary of a quantitative/continuous distribution of sex. Attempts to order categories of DSD into some continuous distribution are doomed to fail—entirely reasonably—if one cannot order

basic properties like sex chromosome conformation or gonad type within a continuous distribution (for example, Montanez, 2017).

Myth: sex is a social construct The spider's web of arguments touched upon here—and including the occasional reminder that sex development is very complicated (Sun, 2019), as if scientists are not well-trained in dissecting complexity to understand fundamental principles—culminates with the premise that the biological categories of sex are constructed by humans. Butler (1990) writes, 'Perhaps this construct called 'sex' is as culturally constructed as gender.' While it is true that scientists observing the natural world develop language and models to describe the natural world, one cannot credibly argue that the phenomena themselves are constructed by humans. If that were the case, not only have humans invented sex but they have also invented stars, gold, clouds and penguins. We have seen that sex is a fundamental property of almost all complex life, and its evolutionary existence pre-dates the human capacity to describe it.

The argument that sex is socially- or culturally-constructed settles, then, at the boundaries between sex categories, and the asserted arbitrariness straddling a fuzzy boundary (an important 'proof' that sex is not observed but 'assigned' at birth). However, the assertion that the categories of male and female are arbitrary because some rare individuals may present with ambiguous sexual anatomy is like asserting that the two different sides of a coin are arbitrary because there exists a non-zero probability a coin may land on its edge. The fact that sex may be ambiguous for some does not call everyone's sex into question. The categories described in humans by 'female' and 'male' are stable, functional, and the dividing line has emerged from observation of our (and other) species, not a coin toss.

Myth: biologists have alternative understandings about sex Finally we challenge the premise that some new scientific consensus on sex has emerged. Writing for German news site DW, Sterzik (2021) claims, 'Yet the broad scientific consensus now looks different: sex is a spectrum.' The definitions and understandings of sex we present in the first two sections of this chapter are uncontroversial, appearing in dictionaries, key biology textbooks and medical consensus statements like that issued by the Endocrine Society (Barghava et al., 2021). There is a vast literature which depends, explicitly or implicitly, on these understandings of sex. Searches on the scientific publication database PubMed for "male" [AND] "sperm" or "female" [AND] "egg"—that is, not exhaustive searches—retrieve around 100,000 results each, including numerous and recent publications from Nobel Laureates in Physiology or Medicine, and from a huge array of biological and medical disciplines.

Furthermore, searches (performed on 9th July 2022) for phrases like 'bimodal sex', 'sex is bimodal', 'spectrum of sex', 'sex is a spectrum' or 'sex is a social construct' generates no results in the biological or medical literature, although two close matches for 'sex is a spectrum' are returned. The first is a study of how sex—female or male—affects the spectrum of genetic variations acquired in the X chromosome over a lifespan (Agarwal and Przeworski, 2019). The second is a study of fetal sex—female or male—affects the spectrum of placental conditions experienced during pregnancy (Murji et al, 2012). Neither study demonstrates any confusion—quite the opposite—about the nature of sex, and both exemplify the importance of understanding sex in a clinical setting. Although not an exhaustive search, it seems that claims of a new scientific consensus—or, at minimum, an academic divide amongst biologists—regarding sex are rather overblown. Such claims are simple appeals to authority, absent from the scientific literature and apparently manufactured by public commentators.

CONCLUSIONS

In this chapter, we have seen that the most prevalent mechanism of reproduction in complex species has stabilised on a binary system of differential gamete types, and the subsequent evolution of body types around this binary system. The majority of species, including humans, are composed of individual females and males, defined by reproductive role, describing their contribution of large, energy-rich gametes (like eggs) or small gametes (like sperm), respectively, to the next generation.

In humans, notwithstanding atypical reproductive development, there are two evolved anatomical body types, each corresponding to one of the two reproductive functions. In utero, females and males develop sex-specific primary characteristics pertinent to reproduction, in the first instance the differentiation of gonad type that will direct future female or male function. Gonads—ovaries or testes, determined in humans by genetic mechanisms—are responsible for both the development of mature gametes (eggs or sperm) and, via hormones, the coordinated development of the relevant reproductive system. In adults, male anatomy comprises testicles, internal genital structures like the vas deferens, and an external penis and scrotum. Female anatomy comprises internal ovaries, internal genital structures like a uterus and vagina, and an external vulva incorporating the clitoris.

Finally, we have dissected arguments that attempt to challenge these basic understandings of sex. We have revealed that the redefinition of sex from an integrated, anatomical system organised around an evolutionary function to a checklist of human-centred, disaggregated physical characteristics is the foundation on which variability of those physical characteristics (in natural or pathological development) is used to deconstruct sex as a binary system, rendering it a construct of the human mind and, if it suits one's political aims, meaningless. We reject such arguments as purely ideological, with no evidence they are taken seriously in the scientific community, lacking explanatory power, and ultimately spurious. Despite the offered alternative frameworks to describe sex, the foundation that is the binary system shines through, underpinning the bimodal peaks of traits or dictating with which other 'point in multidimensional space' a person can successfully reproduce.

REFERENCES

Agarwal, I. and Przeworski, M. (2019). Signatures of replication timing, recombination, and sex in the spectrum of rare variants on the human X chromosome and autosomes. PNAS, 116(36), pp17916-17924.

Ainsworth, C. (2015). Sex redefined. Nature, 518, pp288–291.

Amnesty. (2018). Intersex awareness day: 5 myths [online]. Available at https://www.amnesty.org/en/latest/news/2018/10/its-intersex-awareness-day-here-are-5-myths-we-need-to-shatter/ [accessed 8th August 2022].

Arboleda, V. A. et al. (2014). DSDs: genetics, underlying pathologies and psychosexual differentiation. Nat Rev Endocrinol, 10(10), pp603-615.

Baresi, M. J. F. and Gilbert, S. F. (2020). Developmental Biology. Oxford: Oxford University Press; www.ncbi.nlm.nih.gov/books/NBK9983

Barghava, A. et al. (2021). Considering Sex as a Biological Variable in Basic and Clinical Studies: An Endocrine Society Scientific Statement. Endocrine Reviews, 42(3), pp219–258.

Becks, L. and Agrawal, A. (2010). Higher rates of sex evolve in spatially heterogeneous environments. Nature, 468(7320), pp89–92.

Bell, G. (1982). The masterpiece of nature: The evolution and genetics of sexuality. Berkeley: University of California Press.

Blackless, M. et al. (2000). How sexually dimorphic are we? Review and synthesis. Am J Hum Biol, 12, pp151–166.
Brusman, L. (2019). Sex isn't binary, and we should stop acting like it is [online]. Available at https://massivesci.com/articles/sex-gender-intersex-transgender-identity-discrimination-title-ix/ [accessed 9th August 2022].
Butler, J. P. (1990). Gender trouble: Feminism and the subversion of identity. Abingdon: Routledge.
Callier, V. (2019). Core Concept: Gene transfers from bacteria and viruses may be shaping complex organisms. PNAS, 116 (28), pp13714-13716.
Carroll, L. (1871). Through the looking-glass and what Alice found there. London: Macmillan Publishers.
Casas, L. et al. (2016). Sex change in clownfish: molecular insights from transcriptome analysis. Sci Rep, 6, pp35461.
Charnov, E. L. (1982). The theory of sex allocation. Princeton: Princeton University Press.
Darwin, C. (1859). On the origin of species by means of natural selection, or, the preservation of favoured races in the struggle for life. London: John Murray Publishing.
Darwin, E. (1800). Phytologia: Or, the philosophy of agriculture and gardening. London: Joseph Johnson Publishing.
Davis, W.M., and Uhrin, M. (1991). It Has Been Said. Perspect Biol Med, 34(4), pp549-552.
Desai, M. M. and Fisher, D. S. (2007). Beneficial mutation–selection balance and the effect of linkage on positive selection. Genetics, 176(3), pp1759-1798.
DSD Families [online resource]. Available at https://dsdfamilies.org [accessed 9th August 2022].
Elsesser, K. (2020). 'The myth of biological sex'. Forbes, June 15th, 2020.
Fausto-Sterling, A. (2000). The five sexes: Revisited. Sciences (New York), 40(4), pp18-23.
Fisher, R. A. (1930). The genetical theory of natural selection. Oxford: Clarendon Press.
Forstater, M. (2021). Sex, gender, and medical data. BMJ, 372, n735.
Fraser, J. A. and Heitman, J. (2003). Fungal mating-type loci. Curr Biol, 13(20), R792-795.
Fu, C. et al. (2019). Genetic and genomic evolution of sexual reproduction: echoes from LECA to the fungal kingdom. Curr Opin Genet Dev, 58-59, pp70–75.
Goldman, B. (2019). Animal magnetism [online]. Available at https://stanmed.stanford.edu/2019spring/brains-hard-wired-recognize-opposite-sex.html [accessed on 9th August 2022].
Greiner, S. et al. (2015). Why are most organelle genomes transmitted maternally? Bioessays, 37(1), pp80–94.
Hearne, S. (2021). Species, Individual, Gender—biology and taxonomy don't deal in black and white [online]. Available at https://www.skeptic.org.uk/2021/03/species-individual-gender-biology-and-taxonomy-dont-deal-in-black-and-white/ [accessed 9th August 2022].
Heggie, V. (2015). 'Nature and sex redefined – we have never been binary'. Guardian, February 19th, 2015.
Hildreth, C. (2022). The gender spectrum: A scientist explains why gender isn't binary [online]. Available at https://cadehildreth.com/gender-spectrum/ [accessed 9th August 2022].
Hilton, E. and Wright, C. (2020). 'The Dangerous Denial of Sex'. Wall Street Journal, February 13th 2020.
Hilton, E. and Wright, C. et al. (2021). The reality of sex. Ir J Med Sci, 190(4), pp1647.
Hu, S. et al. (2008). Early steps of angiosperm–pollinator coevolution. PNAS, 105(1), pp240-245.
Hull, C. L. and Fausto-Sterling, A. (2003). Response to: How sexually dimorphic are we? Review and synthesis. Am J Hum Biol, 15(1), pp112-115.
Jaaskelainen, M. et al. (2010). WNT4 is expressed in human fetal and adult ovaries and its signaling contributes to ovarian cell survival. Mol Cell Endocrinol, 317(1-2), pp106-111.

Kashimada, K. and Koopman, P. (2010). Sry: the master switch in mammalian sex determination. Development, 137(23), pp3921–3930.
Kaul, C. et al. (2011). The gender of face stimuli is represented in multiple regions in the human brain. Front Hum Neurosci, 4, pp238.
Kondrashov, A. S. (1988). Deleterious mutations and the evolution of sexual reproduction. Nature, 336(6198), pp435-440.
Lecluze, E. et al. (2020). Dynamics of the transcriptional landscape during human fetal testis and ovary development. Hum Reprod, 35(5), pp1099-1119.
Lehtonen, J. L. and Parker, G. A. (2014). Gamete competition, gamete limitation, and the evolution of the two sexes. Mol Hum Reprod, 20(12), pp1161-1169.
Lessells, C.M. et al. (2009). The evolutionary origin and maintenance of sperm: selection for a small, motile gamete mating type. In Birkhead, T. R., Hosken, D. J. and Pitnick, S. eds Sperm Biology: An Evolutionary Perspective. London: Academic Press, pp43-67.
Lewin, C. and Herlitz, A. (2002). Sex differences in face recognition--women's faces make the difference. Brain Cogn, 50(1), pp121-128.
Lipke, P. N. and Kurjan, J. (1992). Sexual agglutination in budding yeasts: structure, function, and regulation of adhesion glycoproteins. Microbiol Rev, 56(1), pp180–194.
Maddrell, S. H. (1998). Why are there no insects in the open sea? J Exp Biol, 201(17), pp2461-2464.
Magnusdottir, E. and Surani, M. A. (2014). How to make a primordial germ cell. Development, 141(2), pp245–252.
Mamsen, L.S. et al. (2017). Temporal expression pattern of genes during the period of sex differentiation in human embryonic gonads. Sci Rep, 7, pp15961.
Maynard Smith, J. (1978). The evolution of sex. Cambridge: Cambridge University Press.
McCauley, D. E. and Bailey, M. F. (2009). Recent advances in the study of gynodioecy: the interface of theory and empiricism. Ann Bot, 104(4), pp611-620.
Montanez, A. (2017). Visualizing sex as a spectrum [online]. Available at https://blogs.scientificamerican.com/sa-visual/visualizing-sex-as-a-spectrum/ [accessed 9th August 2022].
Moore, K. L. (1968). The sexual identity of athletes. JAMA, 205(11), pp787-788.
Muller, H. J. (1932). Some genetic aspects of sex. Am Nat, 66, pp118–138.
Muller, H. J. (1964). The relation of recombination to mutational advance. Mutat Res, 106, pp2-9.
Murji, A. et al. (2012). Male sex bias in placental dysfunction. Am J Med Genet A, 158A(4), pp779-783.
Nature. (2018). US proposal for defining gender has no basis in science. Nature, 563, pp5.
Oyarzun, P. A. et al. (2020). Trioecy in the marine mussel Semimytilus algosus (Mollusca, Bivalvia): stable sex ratios across 22 degrees of a latitudinal gradient. Front Mar Sci, 7, pp348.
Parker, G. A. and Lehtonen, J. L. (2014). Gamete evolution and sperm numbers: sperm competition versus sperm limitation. Proc R Soc B, 281, pp20140836.
Pollick, F. E. et al. (2005). Gender recognition from point-light walkers. J Exp Psychol Hum Percept Perform, 31(6), pp1247-1265.
Pubmed [online resource]. Available at https://pubmed.ncbi.nlm.nih.gov [accessed 9th August 2022].
Sax, L. (2002). How common is intersex? A response to Anne Fausto-Sterling. J Sex Res, 39(3), pp174-178.
Scharer, L. (2017). The varied ways of being male and female. Mol Reprod Dev, 84(2), pp94-104.
Sinclair, A. H. et al. (1990). A gene from the human sex-determining region encodes a protein with homology to a conserved DNA-binding motif. Nature, 346, pp240-244.

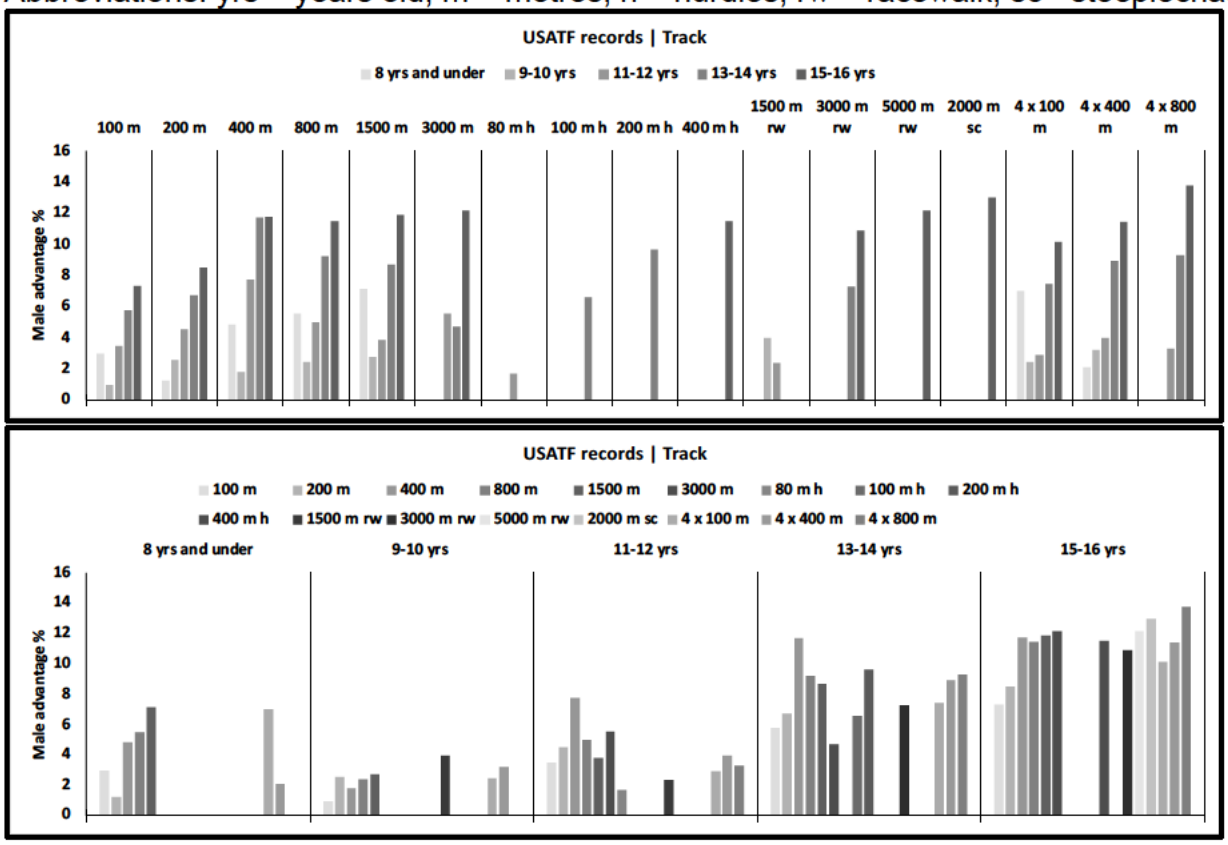
Sterzik, K. (2021). Why sex and gender aren't binary issues [online]. Available at https://www.dw.com/en/why-sex-and-gender-arent-binary-issues/a-57062033 [accessed 9th August 2022].
Sun, S. D. (2019). Stop using phony science to justify transphobia [online]. Available at https://blogs.scientificamerican.com/voices/stop-using-phony-science-to-justify-transphobia/ [accessed 9th August 2022].
van Valen, L. (1973). A new evolutionary law. Evol Theory, 1, pp1–30.
Vonnegut, K. (1969). Slaughterhouse-five, or the children's crusade. New York: Delacorte Publishing.
Weeks, S. C. et al. (2009). Evolutionary transitions among dioecy, androdioecy and hermaphroditism in limnadiid clam shrimp (Branchiopoda: Spinicaudata). J Evol Biol, 22(9), pp1781-1799.
Wizemann, T. M. and Pardue, M. L. (2001). Exploring the Biological Contributions to Human Health: Does Sex Matter? Washington: National Academies Press.
WHO. (2022). WHO updates its widely-used gender mainstreaming manual [online]. Available at https://www.who.int/news/item/06-07-2022-who-updates-widely-used-gender-mainstreaming-manual [accessed 9th August 2022].
WHO. Health topics/Gender [online]. Available at https://www.who.int/europe/health-topics/gender#tab=tab_1 [accessed 9th August 2022].
Wolpert, L. et al. (2019). Principles of Development. Oxford: Oxford University Press.

BIOGRAPHIES

Emma Hilton, Ph.D., is a developmental biologist at the University of Manchester, UK. She has published over 20 manuscripts in development and clinical genetics, and her work on sex-linked genetic disorders has been recognised internationally. In 2021, she published a pioneering review of sports, sex and transgender athlete policy. Colin Wright, Ph. D., is an evolutionary biologist, formerly of Pennsylvania State University, US. Having left academia after a backlash against his public opposition to gender ideology, he now writes as an independent scholar on the biology of sex and sex differences, gender ideology, Critical Social Justice, free speech, and related topics.

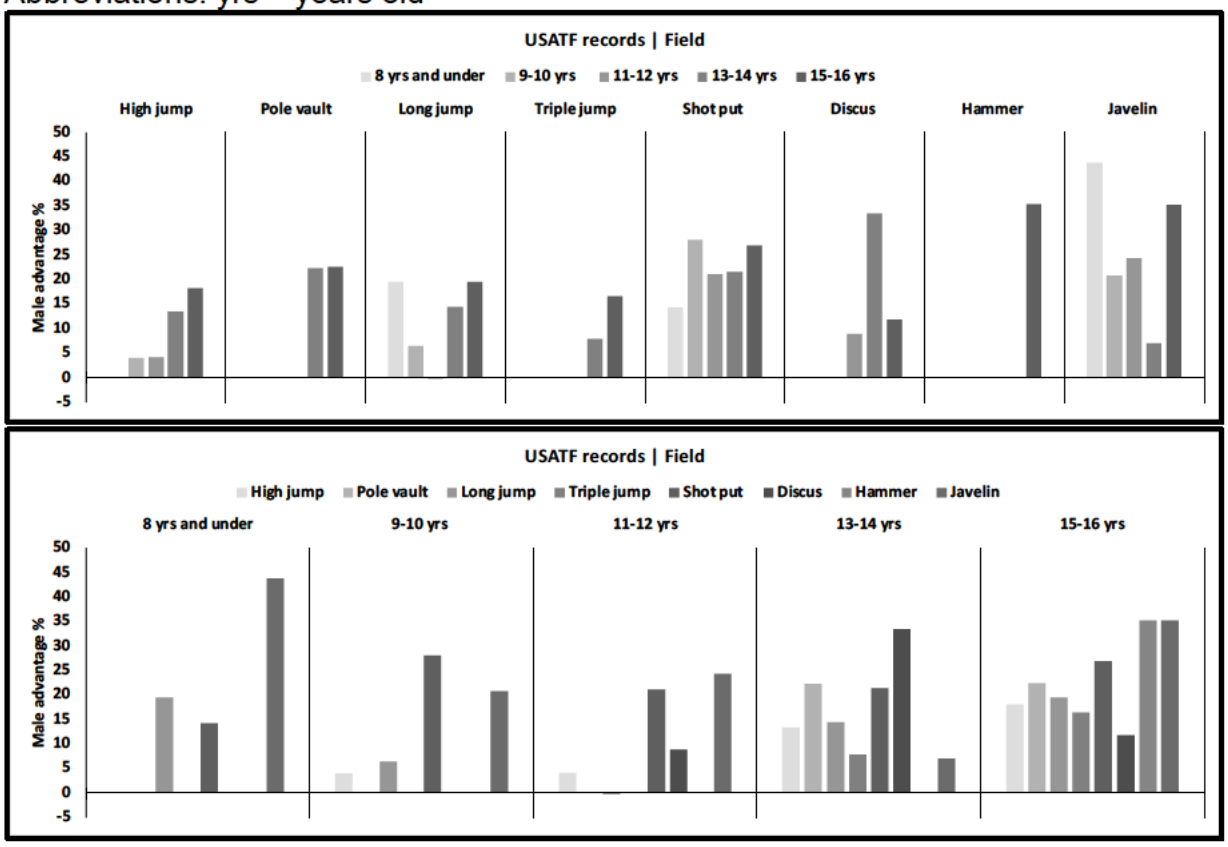
Appendix 3.
USATF junior records from 8-16 years old; analysis of male performance advantage.

Figure A3.1. The male advantage over females in USATF schoolchildren records in track events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc - steeplechase



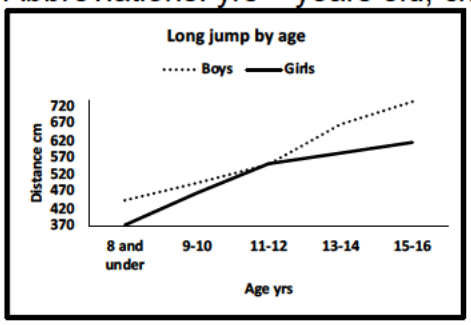
Male advantage is evident in all track events at all ages.

Figure A3.2. The male advantage over females in USATF schoolchildren records in field events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old



Male advantage is evident in all field events at all ages, except long jump/11-12 years old (female advantage 0.2%).

Figure A3.3. Age progression in the long jump in USATF schoolchildren records.
Abbreviations: yrs – years old, cm - centimetres



For long jump at 11-12 years old, female advantage is explained by the convergence of slightly poor male performance and good female performance; perhaps due to pubertal growth spurt in female athlete.

Figure A3.4. Male versus female "wins" in USATF schoolchildren records, scored in track events (upper panel) and field events (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc - steeplechase

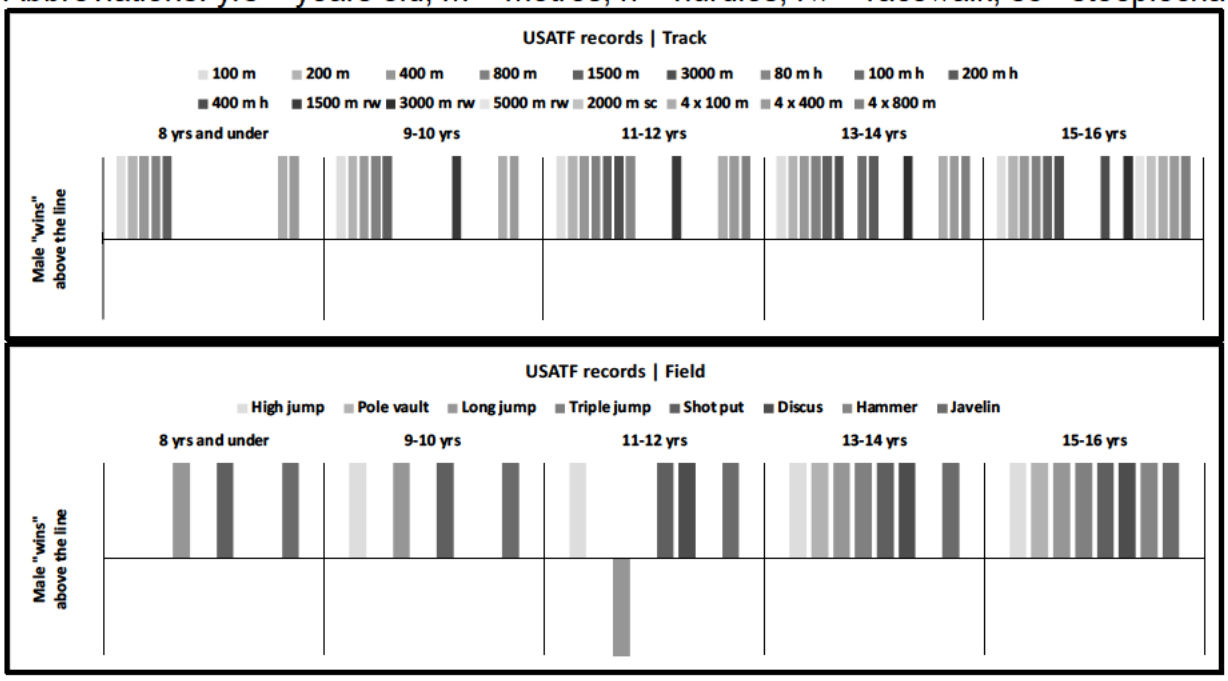
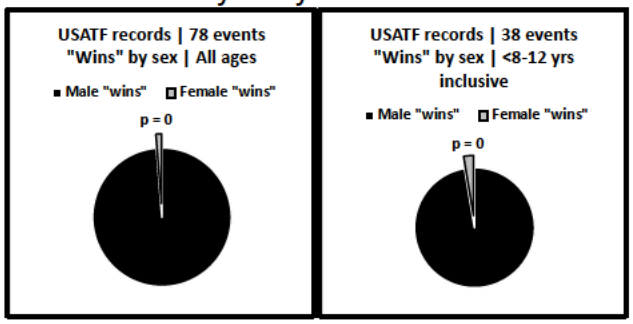


Figure A3.5. The frequency of male versus female "wins" across pooled events in all age groups (left) and limited to pre-puberty age groups (right).
Abbreviations: yrs – years old

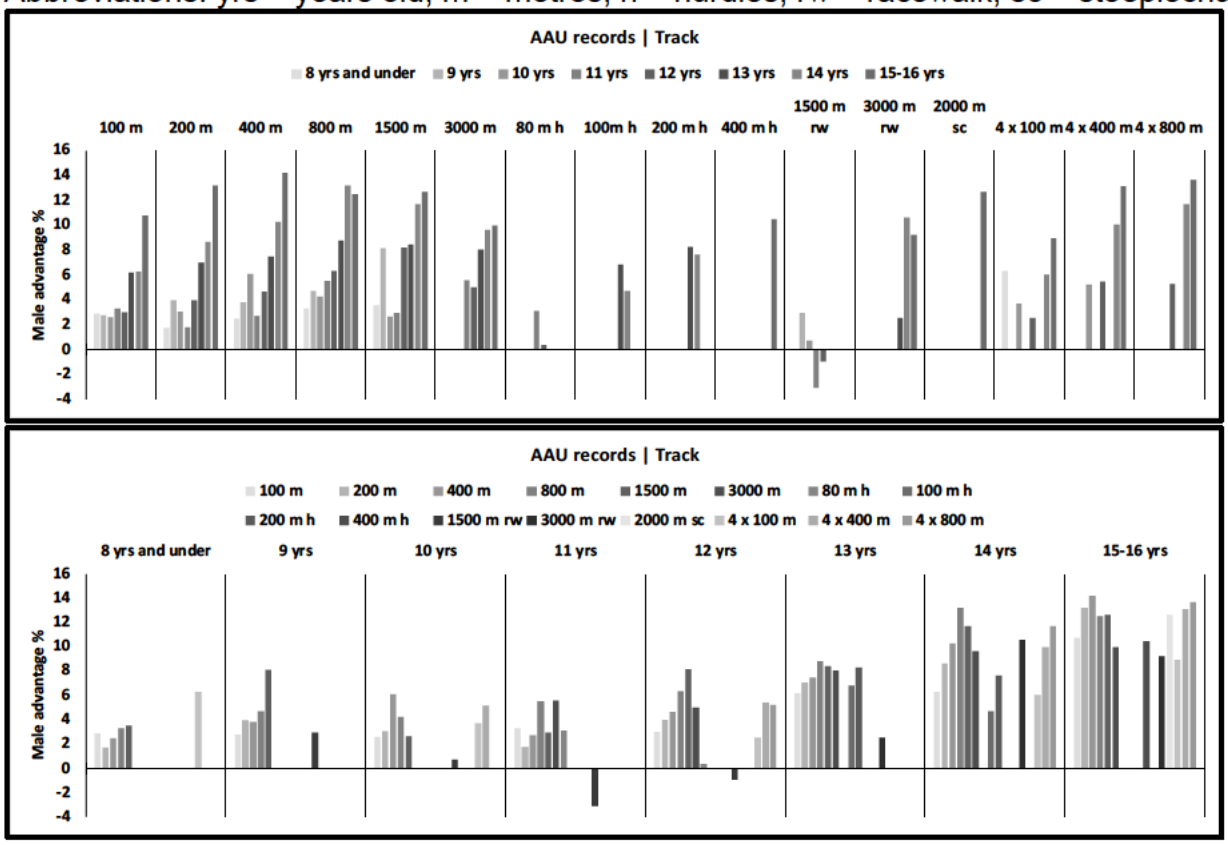


The probability of this frequency of male "wins" occurring by chance, either at all ages or limited to pre-puberty ages, is calculated at as effectively zero (p = 0).

Conclusions from USATF junior record analysis:
1. male advantage over female peers is evident across track and field events from 8 years old onwards.
2. males systematically outperform their female peers from 8 years old, at a frequency that is vanishingly unlikely to result from chance.

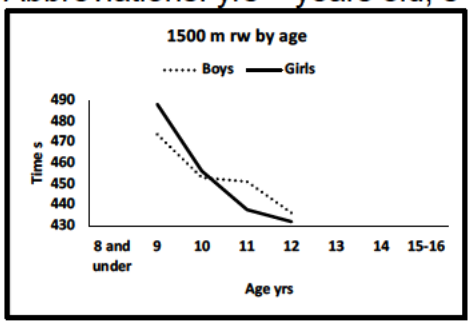
Appendix 4.
AAU junior records from 8-16 years old; analysis of male performance advantage.

Figure A4.1. The male advantage over females in AAU schoolchildren records in track events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc – steeplechase



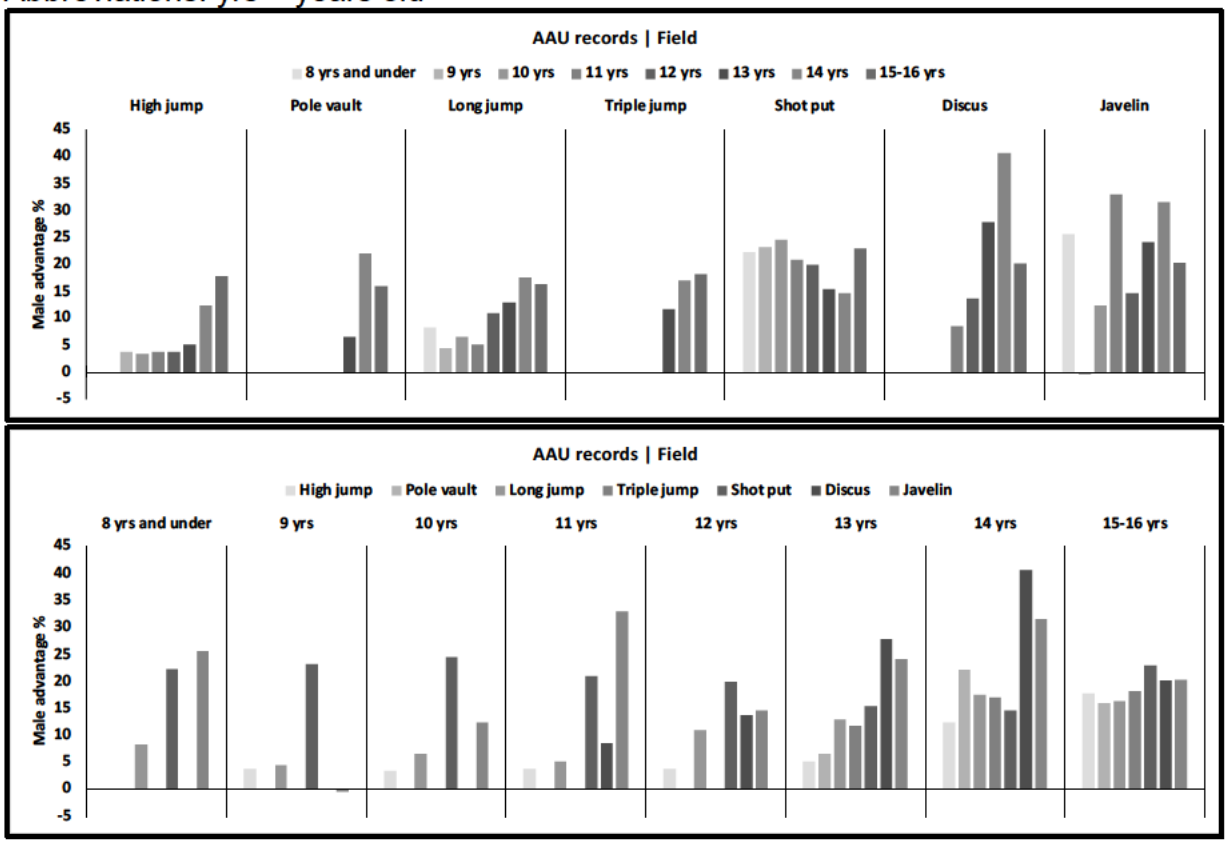
Male advantage is evident in all track events at all ages, except 1500 m rw/11 years old (female advantage 3.1%) and 12 years old (0.9%).

Figure A4.2. Age progression in the 1500 m rw in AAU schoolchildren records.
Abbreviations: yrs – years old, s - seconds



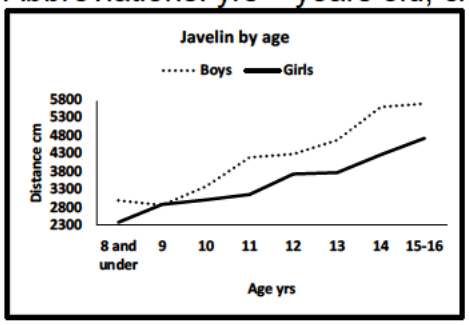
For the 1500 m rw at 11 years old and 12 years old, female advantage is underpinned by good female performances, perhaps explained by pubertal growth spurt synergising with the hip and joint flexibility required for racewalking. This female advantage is transient, and not evident in older age groups in the 3000 m rw event.

Figure A4.3. The male advantage over females in AAU schoolchildren records in field events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old



Male advantage is evident in all field events at all ages, except javelin/9 years old (female advantage 0.4%).

Figure A4.4. Age progression in the javelin in AAU schoolchildren records.
Abbreviations: yrs – years old, cm - centimetres



For javelin at 9 years old, female advantage may be explained by unexpectedly poor male performance converging with good female performance.

Figure A4.5. Male versus female "wins" in AAU schoolchildren records, scored in track events (upper panel) and field events (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc - steeplechase

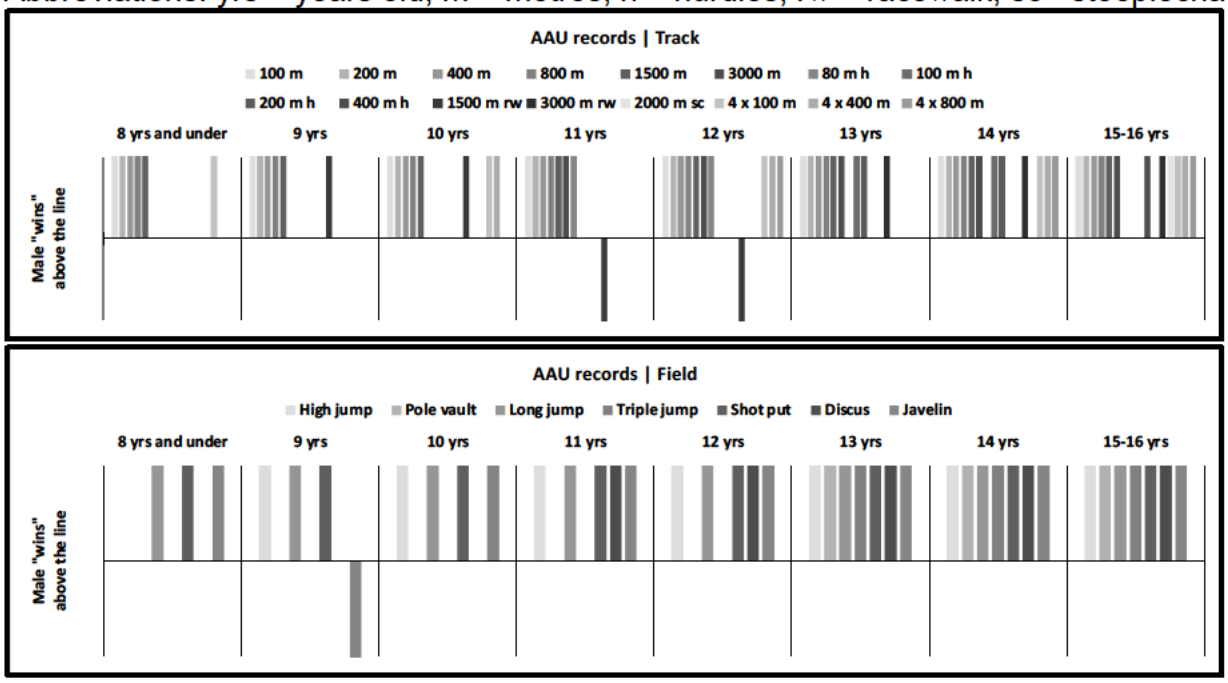
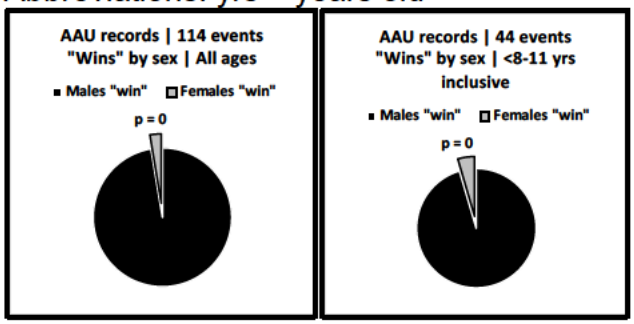


Figure A4.6. The frequency of male versus female "wins" across pooled events in all age groups (left) and limited to pre-puberty age groups (right).
Abbreviations: yrs – years old

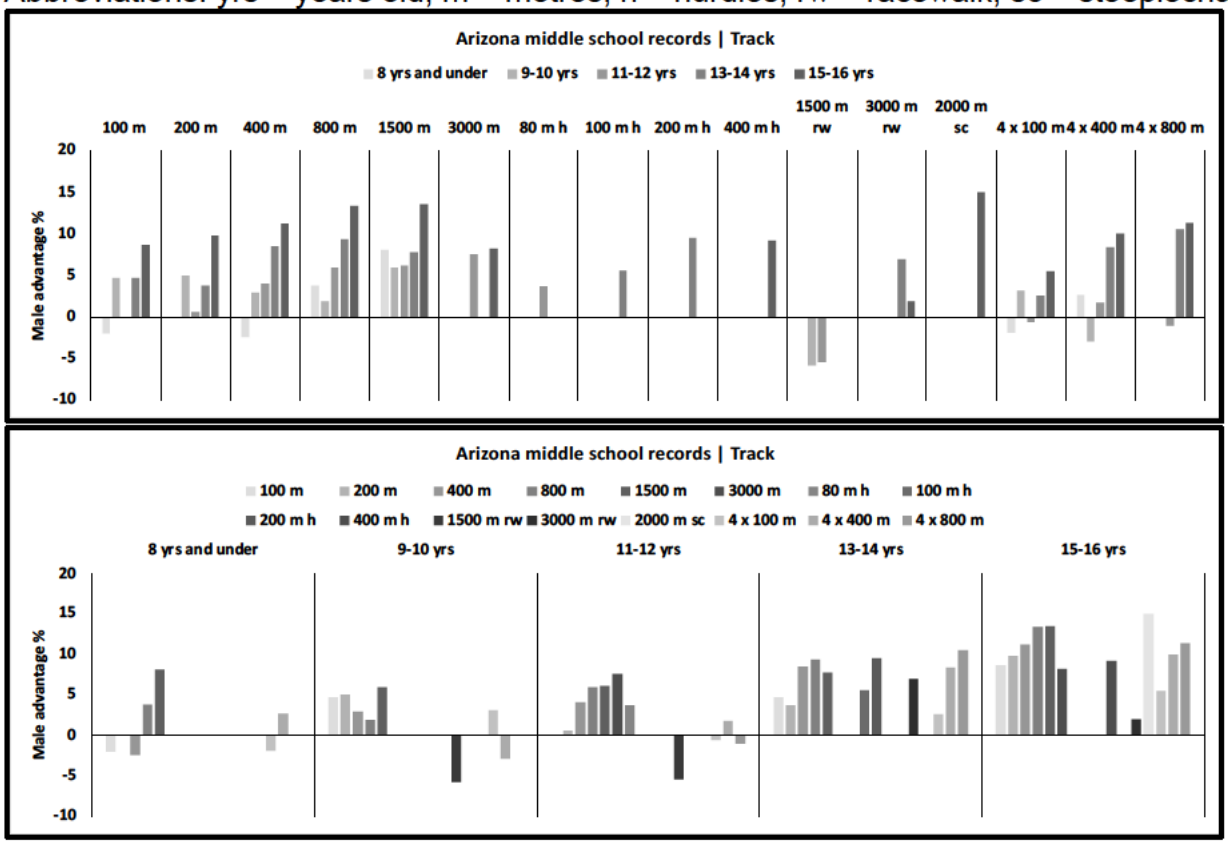


The probability of this frequency of male "wins" occurring by chance, either at all ages or limited to pre-puberty ages, is calculated at as effectively zero (p = 0).

Conclusions from AAU junior record analysis:
1. male advantage over female peers is evident across track and field events from 8 years old onwards.
2. males systematically outperform their female peers from 8 years old, at a frequency that is vanishingly unlikely to result from chance.

Appendix 5.
AZ middle school records from 8-16 years old; analysis of male performance advantage.

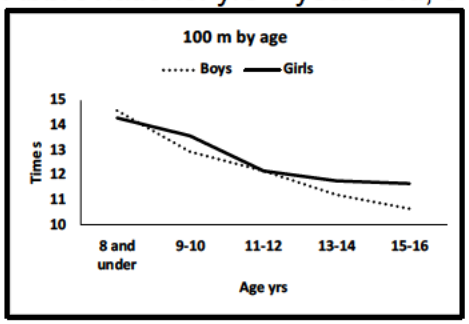
Figure A5.1. The male advantage over females in AZ middle school records in track events, stratified by event (upper panel) and age group (lower panel).
Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc – steeplechase



Male advantage is evident in most track events at all ages. Female advantage was calculated in 9 events:

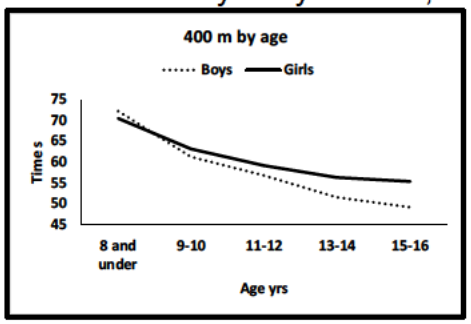
Event	Age group	Female advantage %
100 m	8 yrs and under	2.0 %
	11-12 yrs	0.0 % (same record)
400 m	8 yrs and under	2.4 %
1500 m rw	9-10 yrs	5.8 %
	11-12 yrs	5.4 %
4 x 100 m	8 yrs and under	1.9 %
	11-12 yrs	0.6 %
4 x 400 m	9-10 yrs	2.9 %
4 x 800 m	11-12 yrs	1.1 %

Figure A5.2. Age progression in the 100 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



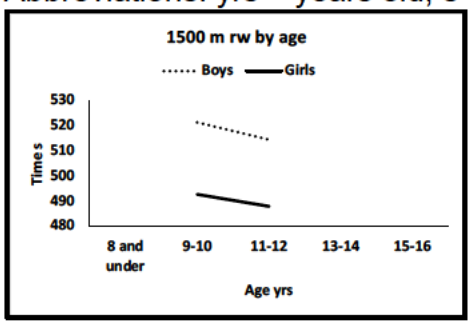
For 100 m at 8 years old and under, in the absence of a preceding datapoint, it is impossible to analyse the apparent female advantage here. The dead heat at 11-12 years old may be explained by good female performance; perhaps due to pubertal growth spurt in female athlete.

Figure A5.3. Age progression in the 400 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



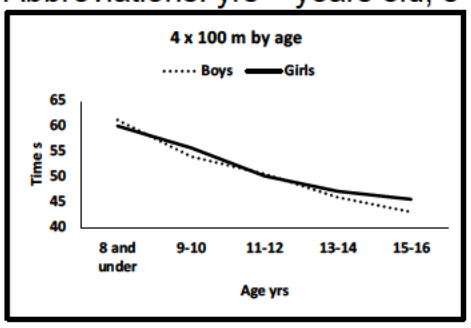
For 400 m at 8 years old and under, in the absence of a preceding datapoint, it is impossible to analyse the apparent female advantage here.

Figure A5.4. Age progression in the 1500 m rw in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



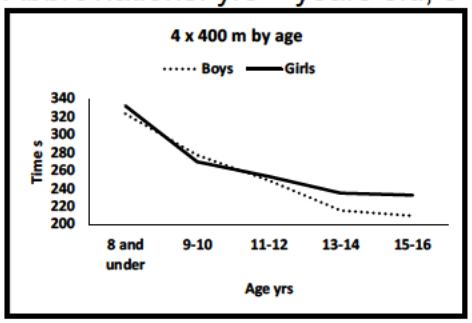
For the 1500 m rw at 9-10 years old and 11-12 years old, female advantage is likely underpinned by good female performances, perhaps explained by pubertal growth spurt synergising with the hip and joint flexibility required for racewalking. This female advantage is transient, and not evident in older age groups in the 3000 m rw event.

Figure A5.5. Age progression in the 4 x 100 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



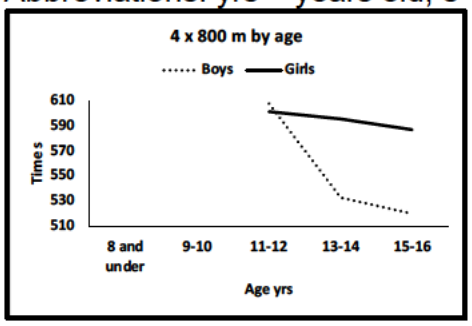
For 4 x 100 m at 8 years old and under, in the absence of a preceding datapoint, it is impossible to analyse the apparent female advantage here. The female advantage at 4 x 100 m/11-12 years old may be explained by good female performance; perhaps due to pubertal growth spurt in females.

Figure A5.6. Age progression in the 4 x 400 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



The female advantage at 4 x 400 m at 9-10 years old may be explained by good female performance; perhaps due to pubertal onset.

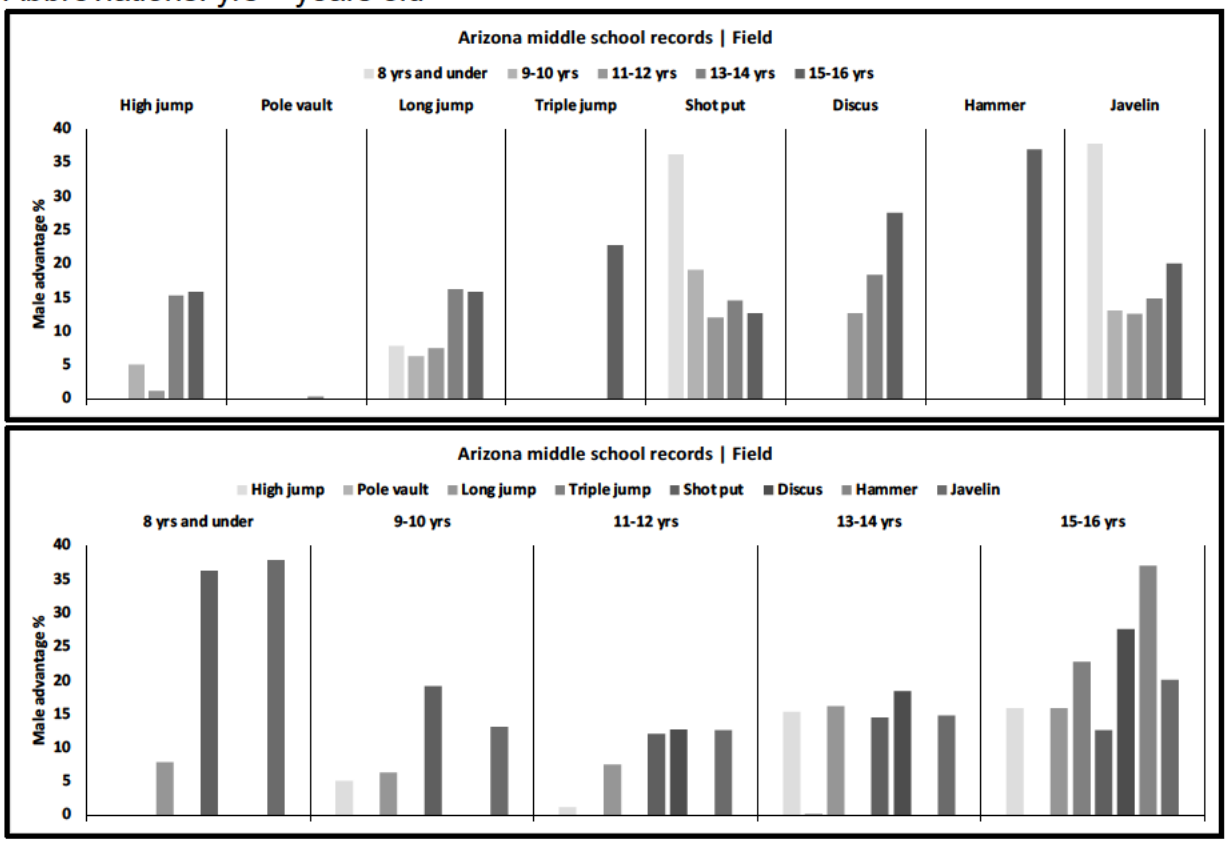
Figure A5.6. Age progression in the 4 x 800 m in AZ middle school records.
Abbreviations: yrs – years old, s - seconds



For 4 x 800 m/11-12 years old and under, in the absence of a preceding datapoint, it is impossible to analyse the apparent female advantage here. However, the unusually-steep male trajectory to 13-14 years old indicates the female advantage at 11-12 years old is likely underpinned by unexpectedly poor male performance.

Figure A5.7. The male advantage over females in AZ middle school records in field events, stratified by event (upper panel) and age group (lower panel).

Abbreviations: yrs – years old



Male advantage is evident in all field events at all ages

Figure A5.8. Male versus female "wins" in AZ middle school records, scored in track events (upper panel) and field events (lower panel).

Abbreviations: yrs – years old, m – metres, h – hurdles, rw – racewalk, sc - steeplechase

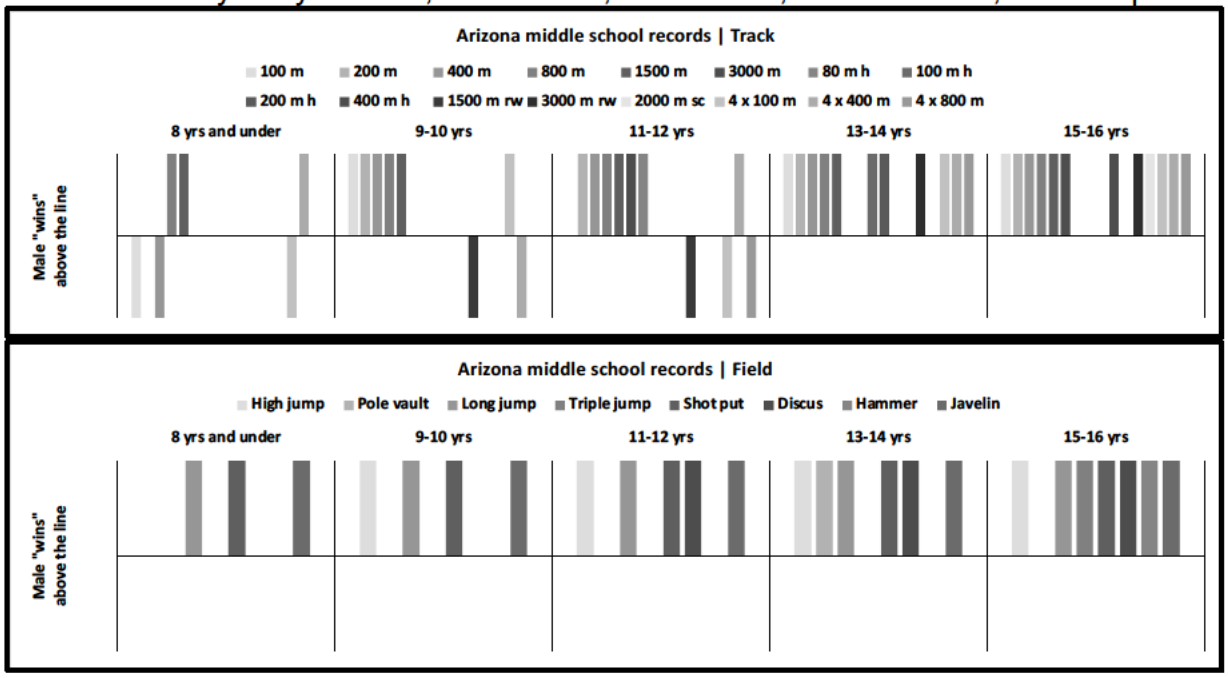
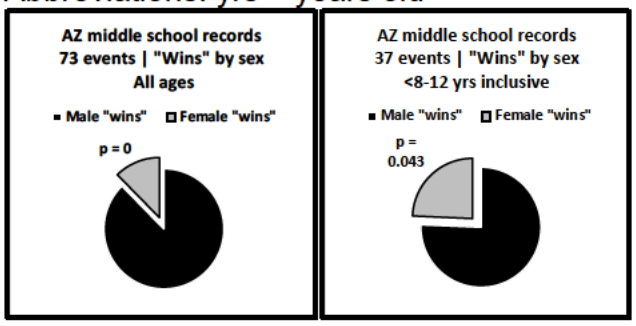


Figure A5.9. The frequency of male versus female "wins" across pooled events in all age groups (left) and limited to pre-puberty age groups (right).
Abbreviations: yrs – years old



The probability of this frequency of male "wins" occurring by chance at all ages is calculated at as effectively zero (p = 0). The probability of this frequency of male "wins" occurring by chance at pre-puberty ages is calculated as p = 0.043, where p > 0.05 represents the threshold of statistical significance in this test. Note: the dead heat in 100 m at 11-12 years old was scored as a female win, to faithfully test the limits of this analysis.

Conclusions from AZ middle school record analysis:
1. male advantage over female peers is evident across track and field events from 8 years old onwards.
2. males systematically outperform their female peers from 8 years old, at a frequency that is unlikely to result from chance.

EXHIBIT 5

WILENCHIK & BARTNESS
A PROFESSIONAL CORPORATION

WILENCHIK & BARTNESS
A PROFESSIONAL CORPORATION

ATTORNEYS AT LAW
The Wilenchik & Bartness Building
2810 North Third Street Phoenix, Arizona 85004

Telephone: 602-606-2810 Facsimile: 602-606-2811

Dennis I. Wilenchik, #005350
Karl Worthington, #018703
admin@wb-law.com

Maria Syms, Bar No. 023019
Director of Legal Services
Arizona Department of Education
1535 W Jefferson, BIN #50
Phoenix, AZ 85007
602-542-5240
Maria.Syms@azed.gov

Attorneys for Defendant Thomas C. Horne

UNITED STATES DISTRICT COURT

DISTRICT OF ARIZONA

Jane Doe, by her next friends and parents Helen Doe and James Doe; and Megan Roe, by her next friends and parents, Kate Roe and Robert Roe, **Plaintiffs,** **v.** **Thomas C. Horne, in his official capacity as State Superintendent of Public Instruction, *et al.*,** **Defendants.**	**Case No. 4:23-cv-00185-JGZ** **DECLARATION OF DR. LINDA BLADE, Ph.D, IN SUPPORT OF DEFENDANT HORNE'S RESPONSE TO PLAINTIFFS' MOTION FOR PRELIMINARY INJUNCTION**

I, Linda Blade, declare as follows:

I submit this expert declaration based upon my personal knowledge.

If called to testify in this matter, I would testify truthfully based on my expert opinion.

QUALIFICATIONS

As a former Canadian Champion (1986) and a full-scholarship NCAA All American (1984) in Track & Field (heptathlon) out of the University of Maryland (1982-1985), I worked hard to be a top student. Academic honors included being named Provost Scholar and member of Phi Beta Kappa.

Now licensed as a Chartered Professional Coach by the Coaches of Canada Association with a PhD in Kinesiology (earned in 1994), I have worked for over 30 years as a "Sport Performance Professional" coaching hundreds of athletes from 5 to 70 years of age, beginner to elite, from many different sports: track & field, hockey, soccer, volleyball, basketball, rugby, triathlon, sailboat racing, football, tennis, squash, swimming, diving, gymnastics, figure skating, skiing and bobsledding.

In my profession as a coach, I blend concepts in human biology with practical coaching methods acquired through many years of personal learning and mentorship opportunities as both athlete and coach. The unique way that I integrate theory and practice has proven to be highly effective. Many top athletes have sought my assistance at various times along their pathway to excellence. At the elite level, I have worked with National Hockey League (NHL) professional players (Edmonton Oilers dryland training, 2016-2018), mentored a world-leading female triathlete (Paula Findlay, 2009-2010) and helped train Pairs Figure Skaters, Jamie Salé and David Pelletier, to an Olympic Gold Medal (2002, Salt Lake City).

Truthfully, though, my greatest accomplishment as a coach has been working with beginners; young athletes ages 6 to 12 years.

It started during my first summer vacation after my freshman year in university. Needing a summer job that would be near the track where I had to continue training, I decided to offer a community "Run, Jump, Throw" camp for kids. Over 200 showed up and seemed to enjoy my coaching. Hosting that camp as a private enterprise became my summer job for consecutive years

of college. I learned how to train children and how to help them improve movement skills that would lay a strong athletic foundation for future success in sports.

Almost a decade after those early years of coaching, my life took an interesting turn. I had finished my PhD in Kinesiology with a subspecialty that focused on measurement of physical growth and development of children (anthropometry), and I was stationed in northern Nigeria (West Africa) at the location that is predominantly Islamic. (This is the same region where the Islamic militant group Boco Haram operates.)

The main university in that region is Bayero University, Kano (BUK). I got my first faculty position there in the Department of Physical Education. Admittedly, it was a bit strange to have a Canadian woman (me) teaching courses, including track and field activity courses, to prospective teachers at one of the top centers of Islamic Studies in Africa.

World Athletics got wind of this situation all the way over in Monaco and suddenly I was recruited (1993) by the CEO of World Athletics' global coaching development, Bjorn Wangemann. His plan was to train and send a world-leading female instructor (me) into Islamic countries to teach women how to coach young girls. There was, of course, a need in religiously segregated places to have female instructors deliver the global coaching certification programs.

This is how I came to be teaching the World Athletics Level 1 (for beginners) coaching curriculum in various countries during the 1990s: in Bahrain, Puerto Rico, Guyana, Kenya, and Sri Lanka.

The highlight of that experience was the course I taught in Iran in July of 1995. I was sent into Tehran to deliver the World Athletics certification course to 30 of the top female coaches selected from across that country. I was the first Western woman since Ayatollah Khomeini's 1979 revolution to travel to Iran for the purpose of engaging women and girls in sport.

For me, personally, that trip to Iran was a wakeup call. I witnessed firsthand what life is like when women & girls are not respected nor given the same rights as men and boys in society. Navigating the "opportunity gaps" in search of training spaces where I could teach the women without male interference was unbelievably challenging. It showed me how vulnerable women's rights can be, including the severely limited access that women can have to their own sporting

experiences. I vowed to never again take such things as Title IX and open access to women's opportunities for granted. I could see that what women in the West have achieve in sports is historically unique and politically fragile.

In 1997 a story about my travels as a global coaching instructor appeared in Sports Illustrated.[i]

Once becoming a mother (1998) and I settled down to a life of coaching in Edmonton, Alberta. Almost immediately, I was approached (1999) by a leading authority in Canadian Track & Field with a special request to author a curriculum piece for basic athletics instruction of children ages 5-11. The timing was perfect. I poured every bit of knowledge I had acquired as top athlete, scholar of child growth, academic instructor, and global coaching lecturer into the Athletics Canada "Run, Jump, Throw" (RJT) program (2001).[ii] Eventually, the rights to that RJT program were purchased by the Hershey's Track and Field Youth Program (2007). A video describing the RJT program can be found here: https://www.youtube.com/watch?v=TQMEg2D0TTw.

More recently, I have authored an update to the RJT program for children called the "Mini Legends Program." [iii]

In 2014, after years of developing children's sports programs and coaching hundreds of athletes at all levels of expertise, I became nominated and voted into office as President of the Board at Athletics Alberta - the track and field association for the province of Alberta. It was while attending national meetings as president in 2018 that I became aware of a philosophy that

seeks to allow male athletes to self-identify into female competitions. I could see in an instant that this would be a catastrophe for female athletes.

Throughout my professional career, I have always maintained that it is unfair for males to compete with females at any age. I believe it is a clear example of discrimination on the basis of sex.

My argument as to why female children should have their own category will now be explained.

REASONS WHY PRE-PUBESCENT GIRLS DESERVE FEMALE-ONLY SPORTS

A few items require clarification before I delve into my rationale.

A. Terminology - For the sake of clarity in my usage of language I will use biological terminology to reflect sex, which is the key determinant of physical reality and performance. For a male-born child I use the word "boy" and pronouns "he/him" (irrespective of social identity). Likewise, for the female-born child I must use the word "girl" and pronouns "she/her."

B. Age delimitation - Since puberty onset can happen as early as nine years of age in some children (especially in girls, who mature on average two years earlier than boys) any comparison of boys and girls deemed to be strictly "pre-pubertal" must be delimited to data obtained at eight years of age and earlier. Therefore, any references I make to data collection and results for prepubertal school children will focus on the 6- to 8-year-old range.

C. Data artifact – In the age range of 9-11 years, due to the phenomenon I mention above, some of the top girls can appear to be "catching up" to the boys in measures of fitness and sport performance. Charts often show a narrowing of the sex differences during this age range. This narrowing of differences between boys and girls is a temporary outlier that arises from the early maturation of a few girls. It is important to note that this phenomenon does not happen for *all* girls at this age range. Therefore, as a coach I will never assume that just because

one of the girls (ages 9-11) outperforms her entire class during a drill that it means I should expect the rest of the girls to be able to perform at the same level.

REASON 1 – Physical

The effect of testosterone on human sexual differentiation is an important factor, albeit not the *only* factor in causing boys to have an advantage over girls in sports. "Sexual dimorphism" (male versus female body design differences) arises from the interaction of testosterone and male genetics encoded by the SRY gene (usually found on the "Y" chromosome). The presence of testosterone in the womb triggers a male baby to begin its journey down the pathway to male morphology. There will be thousands of ways (from the cellular level to the overall anatomy level) in which a male baby diverges in form and physiology from a female baby. Height and weight charts at birth are sex specific, of course.[iv] Key differences in brain circuitry and musculoskeletal features develop before birth and will play a role in providing the male child with advantages related to sport performance. These involve the stitching together of subnetworks in the brain that provide a male child with better movement control, coordination, visual and special awareness, and internal proprioception.[v]

The article cited here mentions that there are differences even in the relative bone lengths of the fingers at birth, with boys having a longer 4th digit (ring finger) relative to the 2nd digit (index finger) and girls having a longer index finger (a larger "D2:D4 ratio"). This seemingly insignificant observation hints at sex-based differentiation in skeleton and joints. As a coach I witness with regularity how little boys have so much more strength in their upper body (upper torso, arms, and shoulders) compared to little girls. This manifests most noticeably when children try to climb or do pull-ups. Indeed, when I look at the data charts included in the *President's Council on Physical Fitness and Sports* (1985)[vi], I see that the sex difference is stark when it

comes to such upper-body performance measures as pull-ups and flexed arm hang. Here is a summary of those data:

Average number of pull-ups at ages 6, 7 and 8:

Boys = 1.3, 1.8, 2.3

Girls = 0.7, 0.8, 1.0

Average time (seconds) a child can maintain the flexed arm hang at ages 6, 7 and 8:

Boys = 7.9, 10.6, 12.3

Girls = 7.1, 9.3, 9.7

The task of gripping a bar and pulling up one's own body weight involves a kind of "leveraging" of forces at the shoulder, upper torso, arms, and hands. In my educated opinion, the sex-based differences in this physical test strongly suggest that the bones and muscles of boys develop differently in structure. The shape of the shoulder joint, the angles of pull, the muscular strength, and durability of that entire set of bony and muscular levers, enables the boys to do so much more.

But, of course, there are differences in other measures, too. Data from the same *President's Council* tests include the following items:

- Mile Run (seconds)
- Long Jump (inches)
- 50 Yard Dash (seconds)
- Shuttle Run (seconds)
- 2 Mile Walk (seconds)
- Sit & Reach (inches)
- Sit-ups (number)

Here are the comparisons by age (highlighted scores are the ones where girls are equal or better):

At AGE 6

	BOYS	GIRLS
Mile Run	788.57	829.21
Long Jump	44.59	40.60
50 Yard Dash	10.22	10.68
Shuttle Run	13.47	13.88
2 Mile Walk	2038.02	2114.23
Sit & Reach	.64	2.43
Sit-ups	22.56	22.90

At AGE 7

	BOYS	GIRLS
Mile Run	726.96	789.73
Long Jump	47.36	43.30
50 Yard Dash	9.82	10.19
Shuttle Run	12.96	13.52
2 Mile Walk	2031.31	2146.35
Sit & Reach	.69	2.23
Sit-ups	27.16	25.37

At AGE 8

	BOYS	**GIRLS**
Mile Run	684.77	763.25
Long Jump	51.83	47.42
50 Yard Dash	9.27	9.71
Shuttle Run	12.39	13.15
2 Mile Walk	1969.93	2078.52
Sit & Reach	.18	2.06
Sit-ups	30.48	28.66

In summary, this testing protocol indicates that boys run faster, have greater endurance, are more agile, jump farther and have greater upper body strength than girls, whereas girls are more flexible (indicated here in the sit and reach test).

This sub-set of results from top finishers at the 2022 AAU National Championship Jr Olympics shows a similar outcome for 8-year-olds[vii]:

	BOYS	**GIRLS**
100m Dash (sec)	13.87	14.41
200m Dash (sec)	28.56	29.64
1500m Run	5:07.14	5:18.44
Long Jump (m)	4.09	3.86
Shot Put	31 ft 1.00 in	23 ft 4.75 in

This chart (above) provides additional evidence that is prototypical. Once again, boys are faster and throw and jump farther than girls. Measurements of lung function in small children - with boys having a higher lung volume[viii], more air passages and other enhanced capacities throughout the oxygen transport system[ix] - explains why they also do better in endurance tests and the 1500m run as reflected in the charts.

I leave it up to other experts like Dr. Gregory Brown and Dr. Emma Hilton, whose reports I have reviewed in preparing my opinion, to provide more such data. The point I wish to make

here is that consistently across all data bases and amongst the the hundreds of children I have worked with as a coach, boys are better than girls in all fitness parameters except in flexibility and, possibly, balance.

In the realm of physical education and sports we refer to human movement capacities as "biomotor abilities." Some coaches say there are only five, but I recognize ten biomotor abilities (with the main physical factors that influence them in brackets):

- Strength (nervous system, muscles, bone structure & joints)
- Speed (nervous system, muscles, bone structure & joints)
- Stamina (cardiovascular system – heart, lungs, blood & cellular substructures)
- Power (nervous system, bone structure, muscles & joint durability)
- Speed-Endurance (cardiovascular system, bone structure, muscles, nervous system & cellular substructures)
- Muscular-Endurance (cardiovascular system, bone structure, muscles, nervous system & cellular substructures)
- Coordination (proprioception, nervous system, muscles & joints)
- Agility (proprioception, nervous system, muscles & joints)
- Balance (proprioception, location of center of gravity, nervous system & muscles)
- Flexibility (softness of joints; extensibility of muscles and ligaments)

And possibly an 11th one that only top coaches talk about (& professionals like NFL quarterback Tom Brady)[x]:

- Elasticity or Pliability (the ability of the entire body or parts of the body to "whip" – to bend and snap like an elastic band)

Due to the underlying structural differences in the nervous system, musculo-skeletal system, and cardio-vascular system, boys have the advantage in nine out of the eleven biomotor abilities.

Girls do excel in sports where flexibility is a dominant feature. For example, boys typically don't compete in rhythmic gymnastics. It requires body contortions that most males are simply

unable to achieve. On the other hand, having hyper-flexible bodies accompanied by lower muscular strength renders girls are highly prone to impact injury in contact sports.

Since most sports involve a combination of biomotor abilities, the male performance advantage will be amplified. In a sport like volleyball, soccer, and basketball where strength, speed, power, endurance, agility, and coordination all come into play, the performance difference compared to the girls will be more obvious than what might be observed in a singular biomotor skill test.

This concept of "additive advantage" is the reason why changing one variable in a boy (say, testosterone level) will not work to fully diminish his performance advantage over his female counterparts. While hormone therapy might diminish a percentage of his original strength and, possibly, endurance, it will not adequately diminish other factors that add up to giving him an overwhelming advantage. For the sake of argument, if boys are better than girls because they are adding up a set of advantages "A + B + C + D + E + F," they will continue to have an advantage even if factor "D" is removed. The male advantage will then be of the set "A + B + C + E + F." It will *still be* insurmountable for the girls.

In summary, as a coach with extensive education in kinesiology – looking at human form and function - I can confirm without hesitation that prepubescent girls as a class will never be able to overcome the performance edge enjoyed by their male cohorts. While not as overwhelming as the differences encountered post-puberty, the sport performance differences enjoyed by pre-pubescent male children are significant and easily recognized by those of us involved: teachers, coaches, parents, and the children. The important point to be made here is that boys will dominate girls in competition because of prepubescent physical differences.

REASON 2 - Psychosocial

As a coach for almost 30 years observing boys and girls in sports competition, I have regularly observed the psychosocial risks of forcing girls to compete against boys. Most little girls simply do not wish to compete against the boys. Girls recognize the categorical difference in biological sex and, as a coach, I have seen quite often that little girls become intimidated when they are compelled to test themselves relative to boys. On a soccer field, a little girl will often

stand back and let the boy take the ball. In games like dodgeball girls will often shy away from the aggressive play of boys. Conversely, when little girls compete with each other their confidence grows and they become far more engaged in the match.

This is the same phenomenon witnessed in girls-only schools. A disadvantage with having to compete with boys is described thus: "In coeducational classrooms, boys tend to monopolise discussion, and take more domineering roles in group work and in practical exercise." [xi] And: "…teachers [and coaches] tend to ignore the strong correlation between high motivation and high anxiety in many high-achieving girls. In girls-only environments, girls' needs and preferences come to the fore."

Based on my observations and interactions with children and families over the course of my 30 years of coaching, I have repeatedly seen that the moment a boy is mixed in with the girls in a highly competitive environment, much of the focus turns to him and his needs at the expense of the girls, who tend to quietly withdraw their assertiveness. Recently, a father told me that his nine-year-old daughter's soccer team had to play against another team that had a male child who "identifies as a girl." He said that the girls on his daughter's team became less energized than usual and did not even try to take the ball away from the boy. Their team ended up losing by many points and the girls left the field asking why they should even be playing. This is the opposite of female empowerment.

Female empowerment takes another huge hit when male children are allowed to share a locker room with the girls. One needs only to hear the testimony of swimmer Riley Gaines to understand the devastation and humiliation involved in dealing with compelled sharing of an intimate space.[xii] It leads to tears and long-lasting psychological distress.

The essence of positive empowerment is what happened when female-only sports exploded in popularity after the passage of Title IX. The numbers don't lie. While there is no data for

primary schools, we can see what happened with older female students, as summarized in this chart:[xiii]

TIME	**MALE** participation in high school sports (number of boys)	**FEMALE** participation in high school sports (number of girls)
Before Title IX [School year 1971-1972]	3,666,917 (93%)	294,015 (7%)
After Title IX [School year 2018-2019]	4,534,758 (57%)	3,402,733 (43%)

These data show a 1,057% increase in female participation in school sports over a 45-year period. A similar increase is reflected in the NCAA data and the point is that never in the annals of world history has there been such a drastic change (improvement!) in the enthusiastic engagement and physical play of female persons.

The impact upon America has been unprecedented. Twenty years after the passage of Title IX (in the 1990s) along came the phenomenon of the "soccer mom" – mothers across America who piled their kids into the minivan determined to get their children into sports. A generation of both boys and girls now owe it to those moms for engaging them in sports and other physically active past times. Based on my observations, this volunteerism has had a positive impact on many children and on the sports associations.

One significant impact of granting girls the opportunity to engage in fair competition and to experience achievement has been on the American economy and the business environment. In clear contrast to the pre-1980s, there are now thousands of women across the USA who start their own businesses and lead companies.

What does this have to do with sports? Consider these facts revealed in an article by Forbes[xiv] magazine reporting on a study of working women undertaken by Ernst & Young:

"The study found that 90% of the women surveyed had played sports either at primary and secondary school, or during university or other tertiary education, with this proportion rising to 96% among C-suite women."

Almost all top female CEOs have had a sports background.

There can be no doubt that access to sport engendered by TitleIX has promoted the kind of self-confidence in America's little girls that has inspired them to grow into adult women pursuing high achievement. The benefit to society has been priceless.

CONCLUSION:

In conclusion, I must say that I am deeply concerned about the future of sports for young girls. We often hear the phrase, "Trans rights are human rights." This is true, but by the same token, "Female rights are human rights." Everyone has rights. But for an activity to be considered a "sport," the fundamental ingredient must be "fairness."

In 2021 when the UK Sport Council's Equality Group (SCEG) released its thorough review of transgender inclusion, it arrived at the following conclusion:

"As a result of what the review found, the guidance concludes that the inclusion of transgender people into female sport cannot be balanced regarding transgender inclusion, fairness and safety in gender-affected sport where there is meaningful competition."[xv]

According to the SCEG report, authorities in sex-affected sports must make a choice: prioritize transgender inclusion or prioritize fairness and safety for the female athlete.

I disagree in one way. I believe that we already have full inclusion in sports. Every human person has a biological sex, even if one wishes to self-identify or express as something different. Therefore, there can be a place for everyone within our sex-based eligibility systems.

Nobody benefits in the long run by mixing sports categories. It is my view that the Save Women's Sports Act preserves fairness in sports for female participants of all identities on the basis of sex, as intended by Title IX.

I swear or affirm under penalty of perjury that the foregoing is true and correct.

Dated: June 28, 2023 Signed: */s/ Dr. Linda Blade, Ph.D*

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[i] https://vault.si.com/vault/1997/08/25/teach-coaching-see-the-world-traveling-to-third-world-countries-to-train-coaches-is-linda-blades-idea-of-a-perfect-summer-vacation

[ii] chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://www.northumberlandsportscouncil.ca/wp-content/uploads/2018/08/Run-Jump-Throw-Resource-.pdf

[iii] https://minilegends.ca/

[iv] https://www.cdc.gov/growthcharts/clinical_charts.htm#Set1

[v] chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9331831/pdf/ijerph-19-09103.pdf (page. 3)

[vi] https://eric.ed.gov/?id=ED291714 (Appendix A, pages 56-57)

[vii] http://image2.aausports.org/sports/athletics/results/2022/jogames/jogamescompleteresults.htm

[viii] https://journals.physiology.org/doi/abs/10.1152/jappl.1962.17.4.601

[ix] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5980468/pdf/EDU-0003-2018.pdf

[x] https://www.youtube.com/watch?v=-SSP_qAUtYI

[xi] https://www.gdst.net/publications/why-and-how-girls-thrive-in-girls-only-schools/

[xii] https://www.dailysignal.com/2023/06/21/riley-gaines-describes-sharing-locker-room-lia-thomas/

[xiii] chrome-extension://efaidnbmnnnibpcajpcglclefindmkaj/https://www.nfhs.org/media/1020205/2017-18_hs_participation_survey.pdf

[xiv] https://www.forbes.com/sites/alanaglass/2013/06/24/ernst-young-studies-the-connection-between-female-executives-and-sports/?sh=7edab51333a2

[xv] https://equalityinsport.org/docs/300921/Guidance%20for%20Transgender%20Inclusion%20in%20Domestic%20Sport%202021.pdf (p. 15)

Colin Proksel (034133)
OSBORN MALEDON, P.A.
2929 North Central Avenue, 21st Floor
Phoenix, Arizona 85012-2793
State Bar No. 034133
Telephone: (602) 640-9000
Facsimile: (602) 640-9050
Email: cproksel@omlaw.com
Attorney for Plaintiffs
Additional counsel listed in signature block

UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF ARIZONA
TUCSON DIVISION

Jane Doe, by her next friend and parents Helen Doe and James Doe; and Megan Roe, by her next friend and parents, Kate Roe and Robert Roe,

Plaintiffs,

v.

Thomas C. Horne in his official capacity as State Superintendent of Public Instruction; Laura Toenjes, in her official capacity as Superintendent of the Kyrene School District; Kyrene School District; The Gregory School; and Arizona Interscholastic Association Inc.,

Defendants.

Case No. 4:23-cv-00185-JGZ

PLAINTIFFS' UPDATED EXHIBIT LIST FOR PLAINTIFFS' MOTION FOR PRELIMINARY INJUNCTION

Plaintiffs submit the following list of exhibits, along with copies of exhibits not already filed on the docket, pursuant to the Court's June 14, 2023 Order (ECF No. 80). Plaintiffs respectfully reserve the right to amend this exhibit list in advance of the hearing.

Ex. No.	Description	Location
1	Declaration of Jane Doe	ECF No. 6
2	Declaration of Helen Doe	ECF No. 7
3	Second Declaration of Helen Doe	ECF No. 78
4	Declaration of Megan Roe	ECF No. 8
5	Declaration of Kate Roe	ECF No. 9
6	Declaration of Stephanie Budge, Ph.D.	ECF No. 4
7	Rebuttal Declaration of Stephanie Budge, Ph.D.	ECF No. 65-1
8	Declaration of Daniel Shumer, M.D., MPH	ECF No. 5

9	Rebuttal Declaration of Daniel Shumer, M.D., MPH	ECF No. 65-2
10	AIA's Constitution, Bylaws, Policies, and Procedures 2022-2023, Transgender Policy	ECF No. 51-1
11	Photographs of the Doe Family (*filed under seal*)	ECF No. 108
12	Photographs of the Roe Family (*filed under seal*)	ECF No. 108
13	Jane Doe's Name Change Court Order (*filed under seal*)	ECF No. 108
14	Megan Roe's Name and Gender Change Court Order (*filed under seal*)	ECF No. 108
15	Jane Doe's Passport (*filed under seal*)	ECF No. 108
16	Megan Roe's Passport (*filed under seal*)	ECF No. 108
17	Consideration of Bills: Hearing on S.B. 1165 Before S. Comm. on Judiciary, Jan. 20, 2022, 55th Leg., 2nd Reg. Sess., 00:08:08–01:30:05 (*filed as a non-electronic exhibit*)	ECF No. 88-1
18	David Handelsman, et al., *Circulating Testosterone as the Hormonal Basis of Sex Differences in Athletic Performance*, 39 Endocrine Revs. 803 (2018)	ECF No. 88-2
19	David Handelsman, *Sex Differences in Athletic Performance Emerge Coinciding with the Onset of Male Puberty*, 87 Clinical Endocrinology 68 (2017)	ECF No. 88-2
20	Jonathon W. Senefeld et al., *Sex Differences in Youth Elite Swimming*, 14 PLOS ONE 1 (2019)	ECF No. 88-2
21	Joanna Harper, *Race Times for Transgender Athletes*, 6 J. Sporting Cultures & Identities 1 (2015)	ECF No. 88-2
22	Marnee McKay & Joshua Burns, *When it Comes to Sport, Boys "Play Like a Girl,"* The Conversation (Aug. 3, 2017), https://theconversation.com/when-it-comes-to-sport-boys-play-like-a-girl-80328	ECF No. 88-3
23	Marnee McKay, et al., *Normative Reference Values for Strength and Flexibility of 1,000 Children and Adults*, Neurology, 88 (1) (2017)	ECF No. 88-3
24	World Rugby Transgender Women's Guidelines (2020), https://www.world.rugby/the-game/player-welfare/guidelines/transgender/women	ECF No. 88-3
25	Governor Douglas A. Ducey's Letter to Arizona Secretary of State re: Senate Bill 1138 and 1165	ECF No. 88-3
26	Second Declaration of Helen Doe	ECF No. 109
27	Second Rebuttal Declaration of Daniel Shumer, M.D., MPH	Attached

Respectfully submitted this 7th day of July, 2023.

/s/ Colin M. Proksel

Colin M. Proksel (034133)
OSBORN MALEDON, P.A.
2929 North Central Avenue, 21st Floor
Phoenix, Arizona 85012-2793
Telephone: (602) 640-9000
Facsimile: (602) 640-9050
Email: cproksel@omlaw.com

Jyotin Hamid*
Justin R. Rassi*
Amy C. Zimmerman*
DEBEVOISE & PLIMPTON LLP
66 Hudson Boulevard
New York, New York 10001
Telephone: (212) 909-6000
Facsimile: (212) 909-6836
Email: jhamid@debevoise.com
Email: jrassi@debevoise.com
Email: azimmerman@debevoise.com

Amy Whelan*
Rachel Berg*
NATIONAL CENTER FOR LESBIAN RIGHTS
870 Market Street, Suite 370
San Francisco, California 94102
Telephone: (415) 343-7679
Facsimile: (415) 392-8442
Email: awhelan@nclrights.org
Email: rberg@nclrights.org

**Admitted pro hac vice.*

Exhibit 27

Colin Proksel (034133)
OSBORN MALEDON, P.A.
2929 North Central Avenue, 21st Floor
Phoenix, Arizona 85012-2793
State Bar No. 034133
Telephone: (602) 640-9000
Facsimile: (602) 640-9050
Email: cproksel@omlaw.com
Attorney for Plaintiffs
Additional counsel listed in signature block

UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF ARIZONA
TUCSON DIVISION

Jane Doe, by her next friend and parents Helen Doe and James Doe; and Megan Roe, by her next friend and parents, Kate Roe and Robert Roe,

Plaintiffs,

v.

Thomas C. Horne in his official capacity as State Superintendent of Public Instruction; Laura Toenjes, in her official capacity as Superintendent of the Kyrene School District; Kyrene School District; The Gregory School; and Arizona Interscholastic Association Inc.,

Defendants.

Case No. 4:23-cv-00185-JGZ

SECOND REBUTTAL DECLARATION OF DANIEL SHUMER, M.D., IN FURTHER SUPPORT OF MOTION FOR PRELIMINARY INJUNCTION

I, Daniel Shumer, declare as follows:

1. I submit this expert declaration based on my personal knowledge.

2. If called to testify, I would testify truthfully based on my expert opinion.

3. In preparing this declaration, I reviewed the expert declarations submitted by Dr. Emma Hilton ("Hilton Decl.") and Dr. Linda Blade ("Blade Decl.") in support of Defendant Horne's Opposition to Plaintiffs' Motion for Preliminary Injunction. I also reviewed the rebuttal declarations by Dr. Gregory Brown ("Brown Rebuttal Decl."), Dr. Chad Carlson ("Carlson Rebuttal Decl."), and Dr. James Cantor ("Cantor Rebuttal Decl.") that the Intervenors submitted in support of their Opposition to Plaintiffs' Motion for Preliminary Injunction. As with my prior expert declaration, I relied on my scientific education and training, my research experience, and my knowledge of the scientific literature in the pertinent fields. The materials I have relied on in preparing this declaration are the same types of materials that experts in my field of study regularly rely on when forming opinions on these subjects. I may wish to supplement these opinions or the bases for them as a result of new scientific research or publications or in response to statements and issues that may arise in my area of expertise.

Dr. Hilton's Declaration

I. There Is No Evidence Linking In Utero Development or Minipuberty to Athletic Performance and No Credible Medical Reason to Posit Any Such Connection.

4. There is no scientific basis for Dr. Hilton's claim that boys gain an athletic advantage over girls based on exposure to testosterone in utero or during minipuberty. (Hilton Decl. ¶¶ 5.3–5.5.)

5. In a male fetus, testosterone production peaks around 11–14 weeks of gestation (in the first trimester of pregnancy), then declines until it is completely suppressed at birth. Testosterone is necessary during this time for normal development of the genitals. *See, e.g.*, Marianne Becker & Volker Hesse, *Minipuberty: Why Does it*

Happen?, 93 Hormone Rsch. Paediatrics 76 (2020).

6. Male babies also experience an elevation of testosterone after birth, with levels peaking between one to two months old, and returning to prepubertal levels before six months of age. As with the in utero elevation of testosterone, a rise in testosterone during minipuberty correlates positively with growth of the male genitals. *Id.* at 78–79.

7. Contrary to Dr. Hilton's testimony, minipuberty does not result in clinically visible physical changes, other than a possible transient increase in testicular volume.

8. In fact, although Dr. Hilton cites Becker & Hesse's article for the proposition that testosterone levels cause an increase in babies' growth velocity and body weight (Hilton Decl. ¶ 5.5), the article describes the opposite. Becker & Hesse found that testosterone and luteinizing hormone (the hormone that stimulates testosterone production) concentrations "during minipuberty correlate *negatively* with body weight and body mass index [BMI] until the age of 6 years." *Id.* at 80 (emphasis added). A negative correlation between testosterone level and body weight or BMI contradicts Dr. Hilton's assertion that minipuberty in males causes competitive athletic advantage later in life. In addition, the article found that "[d]ata on the influence of minipuberty on growth velocity are conflicting." *Id.*

9. No research has linked this brief exposure to elevated testosterone during minipuberty to any lasting physiological impact, much less to an increase in athletic ability. Nor is there any credible medical basis even to hypothesize such an impact.

II. There Also Is No Evidence Linking Gene Expression to Athletic Performance and No Credible Medical Reason to Posit Any Such Connection.

10. There also is no scientific basis for Dr. Hilton's speculation that boys gain an athletic advantage over girls based on sex-specific genetic architecture that results in approximately 6,500 differences in gene expression. (Hilton Decl. ¶ 5.2.) Dr. Hilton fails to cite any research to connect any differences in gene expression between the sexes to the purported athletic advantage of transgender girls who do not undergo male puberty.

11. Contrary to Dr. Hilton's testimony and as I have previously discussed, there is an overwhelming scientific consensus that the biological cause of average differences in athletic performance between men and women is the rise in circulating levels of testosterone beginning in endogenous male puberty. As Handelsman states, "evidence makes it highly likely that the sex difference in circulating testosterone of adults explains most, if not all, of the sex differences in sporting performance." *See* David J. Handelsman et al., *Circulating Testosterone as the Hormonal Basis of Sex Differences in Athletic Performance*, 39 Endocrine Revs. 803, 823 (2018) (summarizing evidence rejecting the hypothesis that physiological characteristics are driven by the Y chromosome).

III. Any Height Differences Among Male and Female Babies Are Negligible and, in Any Event, Largely Disappear Around the Age of Six or Seven.

12. Dr. Hilton's claim that growth charts reveal that "[m]ales are consistently 1-2 cm taller than females between 0-10 years old" (Hilton Decl. ¶ 4.4) is false.

13. Growth charts show that babies' heights are heavily overlapped, with only negligible differences between boys and girls, which differences almost disappear around 6 to 8 years of age, and do not begin diverging again until puberty (see attached full growth charts at **Exhibit A**):

6– 36 months old:

	6 Months		24 Months		36 Months	
Percentile	Boys	Girls	Boys	Girls	Boys	Girls
95th	72 cm	69.5 cm	93 cm	91.5 cm	102.5 cm	101.25 cm
50th	**67 cm**	**65.25 cm**	**87.25 cm**	**86 cm**	**95.75 cm**	**94.75 cm**
5th	63 cm	61 cm	81.5 cm	80 cm	89.75 cm	88.25 cm

7–12 years old:

	7 Years		8 Years		12 Years	
Percentile	Boys	Girls	Boys	Girls	Boys	Girls
95th	130.75 cm	130.75 cm	137.5 cm	137.75 cm	161.5	163 cm
50th	**121.5 cm**	**121.5 cm**	**128 cm**	**128 cm**	**149 cm**	**151 cm**
5th	113 cm	113 cm	118.5 cm	118.25 cm	137 cm	139 cm

14. The numbers begin to diverge again after around 10 years of age, with girls overtaking males in height and weight for a few years because they typically go through the puberty-related growth spurt around two years earlier than males. *See* Charles Brook, *Mechanism of Puberty*, 3 Hormone Rsch. 52, 53 (1999).

15. Moreover, while post-pubertal boys are taller, on average, than post-pubertal girls, the height ranges for boys and girls continue to be overlapping. Ctrs. for Disease Control & Prevention, *Clinical Growth Charts: Children 2 to 20 Years (5th–95th Percentile)*, https://www.cdc.gov/growthcharts/clinical_charts.htm.

IV. There Is No Evidence That Prepubertal Boys Have a Biological Athletic Advantage Over Prepubertal Girls.

16. Contrary to Dr. Hilton's testimony and as I discussed in my prior declarations in this case, there is a well-established scientific consensus that, before puberty, there are no significant differences in athletic performance between boys and girls. *See, e.g.*, Marnee McKay & Joshua Burns, *When it Comes to Sport, Boys "Play Like a Girl"*, The Conversation (Aug. 3, 2017), https://theconversation.com/when-it-comes-to-sport-boys-play-like-a-girl-80328 (discussing results of research published in American Academy of Neurology Journal).

17. While some studies have found small differences between the performance

of boys and girls with respect to some discrete activities, these studies did not control for other factors, particularly age, location, or athletic experience or exposure. *Id.*

18. When research has controlled for those factors by using representative data, researchers have found that "[a]cross all measures of physical performance, there was one consistent finding. There was no statistical difference in the capabilities of girls and boys until high-school age (commonly age 12)." *Id.* These tests included long jump, muscle strength, walking, jumping, and balancing. *Id.*

19. This finding has been replicated in many other studies, and there is a clear scientific consensus that athletic ability does not diverge significantly until puberty. *See, e.g.*, David J. Handelsman, *Sex Differences in Athletic Performance Emerge Coinciding with the Onset of Male Puberty*, 87 Clin. Endocrinol. 68, 70–71 (2017) ("The gender divergence in athletic performance begins at the age of 12-13 years"); Jonathon W. Senefeld et al., *Sex Differences in Youth Elite Swimming*, 14 PLoS ONE 1, 1–2 (2019) (studying child and youth swimmers and concluding that the data suggests "girls are faster, or at least not slower, than boys prior to the performance-enhancing effects of puberty").

20. In support of her contention that boys have at least some biological advantages in athletic performance over girls before puberty, Dr. Hilton relies primarily on data from physical fitness tests or international track and field event records. The data Dr. Hilton relies on in fact shows several areas where pre-pubertal girls outperform pre-pubertal boys. (Hilton Decl. ¶¶ 7.6, 7.9.)

21. Otherwise, the data Dr. Hilton relies on shows that there is a small difference in performance between prepubertal non-transgender boys and prepubertal non-transgender girls.[1] This data merely observes phenomena across a population sample in isolated areas and does not determine a cause for whatever is observed. There is no

[1] Two of the studies cited by Dr. Hilton are also cited in paragraph 6 of the legislative findings of Arizona's statute. *See* S.B. 1165, 55th Leg., 2d Reg. Sess. (Ariz. 2022), § 6.

reliable basis for Dr. Hilton to attribute those small differences to physiology or anatomy instead of other factors, such as greater societal encouragement of athleticism in boys, greater opportunities for boys to play sports, or different preferences of the boys and girls surveyed. David J. Handelsman, *Sex Differences in Athletic Performance Emerge Coinciding with the Onset of Male Puberty*, 87 Clin. Endocrinol. 68 (2017).

22. Dr. Hilton's statement that the "performance gap in international and national track and field records evident before puberty, somewhat controls for this sociali[z]ation effect, given that one might expect engaged sporty girls to be as well-trained as their male peers" (Hilton Decl. ¶ 7.22) is pure conjecture and lacks any reliable factual basis to support it.

23. Dr. Hilton also discusses the outcomes of two individual middle school track and field competitions held at the Kyrene Aprende Middle School in the last year. (Hilton Decl. ¶¶ 7.17–7.20.) It is my understanding from Plaintiffs' counsel that one of the Plaintiffs in this case will begin attending Kyrene Aprende Middle School this month and that she wishes to participate and compete on the girls' cross-country, soccer, and basketball teams, not the track and field team. Moreover, given the age ranges of the children who attend middle school, this data likely includes some males who have undergone male puberty. It is my understanding from Plaintiffs' counsel that the Plaintiff who will be attending Kyrene Aprende Middle School will not undergo male puberty because she will be taking puberty suppressing medication, which I have discussed in more detail in my prior declarations in this case. Therefore, this data is not relevant to this litigation.

24. In any event, as previously discussed, this data does not determine a cause for the observed differences. Even if this data included only prepubertal boys and girls, there is no reliable basis for Dr. Hilton to attribute the differences observed to physiology or anatomy instead of other factors, such as greater societal encouragement of athleticism in boys, greater opportunities for boys to play sports, or different preferences of the boys

and girls surveyed.

V. Transgender Girls Who Receive Puberty Suppressing Medication at the Onset of Puberty Have No Athletic Advantage Over Other Girls.

25. Dr. Hilton incorrectly asserts that the administration of puberty suppressing medication (also sometimes referred to as puberty blocking medication) to transgender girls does not eliminate the athletic advantage that men and adolescent boys have over women and adolescent girls.[2] (Hilton Decl. ¶ 9.5.)

26. As I have discussed previously, Tanner staging (also called Sexual Maturity Rating) is used to document and track the development and sequence of secondary sex characteristics of children during puberty. Under current standards of care, transgender adolescents are eligible to receive puberty blockers when they reach Tanner Stage 2, at the first onset of puberty, and long before the development of increased muscle mass and strength associated with later stages of male puberty. *See* Wylie C. Hembree et al., *Endocrine Treatment of Gender-Dysphoric/Gender-Incongruent Persons: An Endocrine Society Clinical Practice Guideline*, 102 J. Clinical Endocrinology & Metabolism 3869–903 (2017).

27. Following the administration of puberty blockers, transgender girls will also receive hormone replacement therapy to allow them to go through puberty consistent with their female gender identity. As a result, these transgender girls will develop many of the same physiological and anatomical characteristics of non-transgender girls, including bone size, skeletal structure, and distinctive aspects of the female pelvis geometry that cut against athletic performance. Thus, a transgender girl who received

[2] Dr. Hilton also briefly discusses the medical treatment of transgender girls and states that many children reporting gender dysphoria desist and that puberty blocking medication is harmful and has uncertain outcomes. (Hilton Decl. ¶¶ 9.3-9.4.) These conclusions are contrary to my experience treating over 600 patients with gender dysphoria. Dr. Hilton is not a medical doctor or mental health professional nor does it appear that she has ever treated a transgender patient. Moreover, Dr. Hilton does not explain how any of her criticisms are relevant to the issue of whether transgender girls should be able to participate on female sports teams. In any event, as discussed in detail in my prior declarations in this case, these criticisms are not well-founded.

puberty suppressing medication followed by hormone replacement therapy does not have the same physiology as a prepubertal non-transgender boy.

28. Because such girls do not undergo male puberty, they do not gain the increased muscle mass or strength that accounts for why post-pubertal boys as a group have an advantage over post-pubertal girls as a group.

29. For that reason, studies on transgender women who have undergone testosterone suppression as adults are almost meaningless when assessing the athletic abilities of transgender girls who have received pubertal suppression beginning at the onset of puberty. The women in those studies did not transition until well after puberty and experienced exposure to testosterone over an extended time, allowing their muscles to keep developing. In sharp contrast, transgender girls who receive Gonadotropin-releasing hormone agonist ("GnRHa") do not go through male puberty and are not exposed to the heightened level of testosterone associated with male puberty.

30. Even so, those studies of adult transgender women show that testosterone suppression resulted in significant mitigation of muscle mass and development in adult transgender women.

31. For example, the only study directly examining the effects of hormone therapy on the athletic performance of transgender female athletes is a small study of eight long-distance runners. The study showed that after undergoing medical interventions, which included lowering their testosterone levels, the athletes' performance had reduced so that relative to non-transgender women their performance was now proportionally the same as it had been relative to non-transgender men prior to any medical treatment. In other words, a transgender woman who performed at about 80% as well as the best performer among men of that age before transition would also perform at about 80% as well as the best performer among women of that age after transition. *See* Joanna Harper, *Race Times for Transgender Athletes*, 6 J. Sporting

Cultures & Identities 1 (2015).[3] Given that adolescent transgender girls who receive puberty suppressing medication do not go through male puberty, there is no medical basis to expect that transgender girls receiving such medications would have an athletic advantage.

32. Dr. Hilton cites two studies that she claims show that transgender girls have an athletic advantage over other girls even when they are receiving puberty blocking medication or hormone therapy; however, neither study supports Dr. Hilton's claim.

33. Dr. Hilton cites to Maartje Klaver et al., *Early Hormonal Treatment Affects Body Composition and Body Shape in Young Transgender Adolescents*, 15 J. Sexual Med. 251 (2018). (Hilton Decl. ¶ 11.3.) Contrary to Dr. Hilton's claim, however, the primary finding of the Klaver study is that receiving puberty blockers and hormone therapy bring the body composition of young transgender women much closer to their non-transgender female peers than their non-transgender male peers. Those results are more pronounced the earlier a transgender girl starts puberty blockers. *Id*. at 255 (finding that "compared with adult transgender persons treated with CHT, larger changes in body shape and body composition are seen in transgender persons who start in adolescence"). It should also be noted that the transgender women participants in the Klaver study started GnRHa at an average age of 14.5 years, and none started prior to age 12. This is because the original Dutch protocol did not provide GnRHa prior to age 12 regardless of whether puberty started at a younger age. The participants in the study by definition had much more testosterone exposure than transgender girls treated with modern protocols,

[3] The legislative findings of the Arizona statute incorrectly state that for transgender women who go through male puberty (unlike the plaintiffs here), the benefit conferred by testosterone "is not diminished through the use of testosterone suppression." *See* S.B. 1165, 55th Leg., 2d Reg. Sess. (Ariz. 2022), § 13. While that statement conflicts with available evidence, which shows that hormone therapy significantly reduces muscle mass and strength, it is also irrelevant to the situation of the plaintiffs in this case who have not undergone male puberty and thus are not in the position of having to mitigate the increased muscle mass and strength caused by male puberty. Notably, the legislative findings do not state that transgender girls who receive puberty suppressing medication at the onset of puberty have any conceivable athletic advantage, nor do they cite any evidence that would support that claim.

which initiate GnRHa based on pubertal stage unrelated to age.

34. Dr. Hilton also cites Lloyd J.W. Tack et al., *Proandrogenic and Antiandrogenic Progestins in Transgender Youth: Differential Effects on Body Composition and Bone Metabolism*, 103 J. Clinical Endocrinology & Metabolism 2147 (2018), for the proposition that transgender girls who receive medical treatments purportedly maintain greater grip strength than transgender boys. (Hilton Decl. ¶ 11.3.) But the medication administered in this study is not used in the U.S. and does not have nearly the same impact as puberty blockers and hormone therapy for transgender girls or as testosterone for transgender boys. The medications administered to the study participants did not fully block puberty for either transgender girls or transgender boys. Even with this less effective medication, the study found that transgender girls "showed a significant increase in fat mass and decrease in lean mass, resulting in an increased body fat percentage" and did not experience any increase in grip strength. *Id*. at 2153–54. If anything, this study shows that even with a less effective medication, the physiological impact of medically treating transgender girls in adolescence, rather than when they are adults, is profound.

35. At the beginning of her declaration, Dr. Hilton discusses her involvement with the World Rugby Transgender Guidelines. (Hilton Decl. ¶ 1.13.) However, even these guidelines allow transgender girls and women to participate in women's rugby if they did not experience endogenous puberty, stating: "Transgender women who transitioned pre-puberty and have not experienced the biological effects of testosterone during puberty and adolescence can play women's rugby." World Rugby, *Transgender Women Guidelines* (2019), https://www.world.rugby/the-game/player-welfare/guidelines/transgender/women.

36. In sum, there is no evidence that transgender girls on puberty suppression medication or hormone therapy have an athletic advantage over other girls. There are no studies that have documented any such advantage, and there is no medical reason to posit

that any such advantage would exist.

37. In my clinical practice, I have provided medical care to more than 300 adolescent transgender girls. None of the transgender girls I have treated with the above medical interventions appeared to have any athletic advantage over other girls.

VI. From a Medical Perspective, Menstruation Does Not Provide a Basis to Conclude That Transgender Girls Have an Athletic Advantage Over Other Girls.

38. In her declaration, Dr. Hilton claims that female athletes have an athletic disadvantage because they "must typically deal with the effects of the menstrual cycle," which may affect "training capacity and performance," and that, as a result, transgender girls have an athletic advantage because they do not menstruate. (Hilton Decl. ¶ 6.5.) This conclusion does not have a sound medical or scientific basis because not all adolescent girls menstruate or suffer any athletic disadvantage if they do menstruate.

39. For example, girls with certain medical conditions do not menstruate, and some adolescent girls may take birth control to prevent menstruation or for other medical reasons. In addition, not all adolescent girls who do menstruate suffer any adverse impacts on their training capacity or performance.

VII. Permitting Transgender Girls to Play on Girls' Teams Does Not Pose a Safety Risk to Other Girls.

40. In her declaration, Dr. Hilton testifies that transgender girls who play on girls' teams somehow pose a threat to the safety of other girls because, she asserts, girls have "delicate brain structures" that make them more prone to injury. (Hilton Decl. ¶ 6.6.) While research has found that girls suffer more sports-related concussions than boys, the cause of that differential is unknown, including whether it is cultural or biological or both. *See* William T. Tsushima et al., *Incidence and Risk of Concussions in Youth Athletes: Comparisons of Age, Sex, Concussion History, Sport, and Football Position*, 34 Archives Clinical Neuropsych. 60, 66 (2019). In any event, however, there

is no scientific evidence that girls have more "delicate brain structures" than boys. If a researcher were to view an MRI of a human brain, there would be no way to identify whether it was the brain of a male or a female other than average size. Lise Eliot et al., *Dump the "Dimorphism": Comprehensive Synthesis of Human Brain Studies Reveals Few Male-Female Differences Beyond Size*, 125 Neurosci. & Biobehav. Rev. 667, 668 (2021).

41. Some researchers have theorized that girls may suffer more sports-related concussions because, on average, adolescent girls have weaker neck muscles than post-pubertal adolescent boys. *See* Abigail C. Bretzin et al., *Association of Sex with Adolescent Soccer Concussion Incidence and Characteristics*, 4 JAMA Network Open 4, 6 (2021); Ryan T. Tierney et al., *Gender Differences in Head-Neck Segment Dynamic Stabilization During Head Acceleration*, 37 Med. & Sci. Sports & Exercise 272, 272 (2005). If that accounts for girls' higher rates of concussions (which is unknown), transgender girls on puberty blockers or hormone therapy would be at the same or similar risk for such injury as non-transgender girls. There is no evidence, and no medical reason to believe, that their participation on girls' teams would pose any increased threat of such injuries to other girls.

42. More generally, transgender girls do not present any unique safety risks to other girls. Transgender girls' physical characteristics (in terms of height, weight, and strength) overlap with those of other girls. For example, while some transgender girls may be taller than average, so are some non-transgender girls, and many transgender girls are simply average.

43. There is no more reason to exclude a tall transgender girl for safety reasons than there would be to exclude any other girl for that reason. While some transgender girls may (or may not) have larger skeletons than some non-transgender girls, there is no medical reason to conclude that that physical characteristic poses any elevated safety concerns when not accompanied by high levels of testosterone and corresponding skeletal

muscle. After a transgender adolescent suppresses her level of testosterone, there is no inherent medical reason why her physiological characteristics related to athletic performance should be treated differently from the physiological characteristics of other girls.

Dr. Blade's Declaration

44. Dr. Blade is not a medical doctor, nor does it appear that she has ever treated a transgender patient; in contrast, I have experience treating over 600 hundred patients with gender dysphoria. From a medical perspective, the terms "biological sex," "biological male," and "biological female" are imprecise terms that can cause confusion. A person's sex encompasses several different biological attributes, including sex chromosomes, certain genes, gonads, sex hormone levels, internal and external genitalia, other secondary sex characteristics, and gender identity. Those attributes are not always aligned in the same direction. *See* Joshua D. Safer, *Care of Transgender Persons*, 381 N. Engl. J. Med. 2451 (2019).

45. Contrary to Dr. Blade's testimony and as I have previously discussed, there is an overwhelming scientific consensus that the biological cause of average differences in athletic performance between men and women is the rise in circulating levels of testosterone beginning in endogenous male puberty.

46. Dr. Blade discusses data from physical fitness tests in children to demonstrate that transgender girls have an athletic advantage over other girls before puberty. (Blade Decl. at 7–9.) This data merely observes phenomena across a population sample in isolated areas and does not determine a cause for whatever is observed. As I have discussed previously, there is no reliable basis for Dr. Blade to attribute any small differences between boys and girls to physiology or anatomy instead of other factors, such as greater societal encouragement of athleticism in boys, greater opportunities for boys to play sports, or different preferences of the boys and girls surveyed.

47. Dr. Blade also asserts that because prepubertal boys have a greater lung volume and other enhanced capabilities throughout the oxygen transport system, they do better in endurance tests and the 1500m run. (Blade Decl. at 9.) In fact, any such difference between boys and girls is small and has no documented impact on athletic performance. If this small average difference in lung capacity had a significant causal relationship to athletic advantage, we would see significant differences in the athletic performance of prepubescent boys and girls, but we do not.

48. Dr. Blade posits that transgender girls' participation in girls' sports causes psychosocial risks to other girls. (Blade Decl. at 11–12.) Dr. Blade's assertion is based on a misunderstanding regarding transgender girls. As discussed in my prior declarations in this case, a transgender girl is a girl. Moreover, there is no reason to assume a transgender girl's identity would be discernible to other girls, particularly when a transgender girl transitions socially and medically. Lastly, Dr. Blade supports her assertion with mere anecdotes rather than scientific research on the topic. As discussed above, the scientific research demonstrates there is no athletic advantage between transgender girls who have not undergone male puberty and other girls.

Dr. Brown's Rebuttal Declaration

49. Dr. Brown cites a hodge-podge of studies, but none support his view that prepubertal boys have a significant group-based advantage over prepubertal girls, which is contrary to the overwhelming weight of medical evidence and consensus on this issue.

50. For example, although Dr. Brown claims that Handelman's research supports Dr. Brown's position, Handelman himself disagrees, as Dr. Brown concedes.

51. The studies cited by Dr. Brown do not support his thesis for a variety of reasons. First, several of the studies include post-pubertal as well as pubertal children. (*See*, *e.g.*, Brown Rebuttal Decl. ¶¶ 9–10 (citing data that includes children from the ages of 9 to 16).) Second, some of the studies show small physiological differences between prepubertal boys and girls, but do not purport to establish any causal link between those

small differences and athletic ability or establishing only a speculative or hypothetical link. (*See, e.g.*, Brown Rebuttal Decl. ¶ 12 (citing data showing that girls have a slightly higher resting heart rate).) And third, even with respect to those small physiological differences between prepubertal boys and girls, unlike the post-pubertal production of testosterone, those differences exist on an overlapping spectrum. For example, while it is true that there is some evidence that prepubertal boys on average may have slightly less body fat than girls,[4] there are some girls who have less body fat than some boys, and some boys who have more body fat than some girls. In contrast, apart from girls with certain intersex conditions or other health conditions, there are no post-pubertal girls with more testosterone than post-pubertal boys; generally speaking, testosterone levels in post-pubertal boys and girls do not overlap.

52. Notably, Dr. Brown agrees that there is no basis for alleging that minipuberty has any impact on athletic ability. (Brown Rebuttal Decl. ¶ 37 (stating "At no point in my declaration are the male athletic advantages differences ascribed to 'minipuberty' (indeed, the term 'minipuberty' is not found within my expert report.")).

Dr. Carlson's Rebuttal Declaration

53. Dr. Carlson acknowledges that the only studies finding small differences in athletic performance between prepubertal boys and girls are cross-sectional studies that, as such, do not "assign causation to any measured differences, such as biology vs. sociological effect." (Carlson Rebuttal Decl. ¶ 6.) In addition, the small differences found by these studies relate to discrete activities, not to strength or athletic performance across the board, and do not rise anywhere close to the level of the broad, clear, and significant group-based differences caused by exposure over time to the elevated levels of testosterone associated with male puberty.

54. Dr. Carlson attempts to rebut the conclusion of McKay's study that there

[4] As noted in my prior declaration, and as Dr. Brown acknowledges (Brown Rebuttal Decl. ¶ 17), this research is not conclusive; some studies have found no differences and have criticized other studies for failing to consider factors such as age, maturational status and obesity status. (Shumer Rebuttal Decl. ¶ 6)

are no significant differences in athletic ability between prepubertal boys and girls, but his analysis is not persuasive. As Dr. Carlson acknowledges, McKay found no significant differences in strength based on sex in children ages 3 through 9—i.e., in prepubertal children, and found such differences only in post-pubertal children. (Carlson Decl. ¶ 9).

55. Dr. Carlson's suggestion that the two girls who are Plaintiffs in this case would have been grouped with the 10 to 19 year olds (Carlson Rebuttal Decl. ¶¶ 10–11) has no logical relevance to the import of McKay's study: significant athletic differences between boys and girls are linked to puberty. The Plaintiffs in this case are receiving puberty suppressing medication, which prevents them from undergoing male puberty and thus from gaining the potential athletic advantage associated with exposure to post pubertal levels of testosterone.

56. Dr. Carlson acknowledges that the studies he cites "carry with them the limitations of cross-sectional comparisons" (Carlson Rebuttal Decl. ¶ 15), and thus cannot establish any causal link between physiology and athletic performance in prepubertal children for the reasons explained above.

57. Dr. Carlson offers no evidence for his assumption that the enactment of Title IX means that prepubertal boys and girls now receive equal coaching and skill training, nor does any such evidence exist. (Carlson Rebuttal Decl. ¶ 19) To the contrary, as discussed below, research shows that girls receive far less opportunities for participation than boys.

58. Relatedly, Dr. Carlson relies heavily on a single article by Lombardo, which in turn rests upon speculative and subjective hypotheses about how boys and girls are treated in various cultures, including, for example, a presumption that Aboriginal boys and girls are equally encouraged to hunt and that German boys "do not throw much and do not have U.S.-like cultural support or encouragement for throwing." (Carlson Rebuttal Decl. ¶ 19(citing Michael P. Lombardo et al., *On the Evolution of the Sex*

Differences in Throwing: Throwing is a Male Adaptation in Humans, 93 Q.Rev. Biology 91 (2018))). Such speculative research based on broad sociological generalizations about other cultures does not provide a valid evidentiary basis to conclude that the small differences in athletic performance found in some cross-sectional studies of prepubertal boys and girls are based on physiology rather than culture, much less that such small differences have any applicability to individual transgender girls or warrant excluding all transgender girls from playing on girls' teams.

59. Research that is more carefully and objectively designed has found that differences in skills training and practice—not innate gender-based differences—account for many specific sex-based differences in athletic performance. For example, a 2019 study of spatiotemporal coordination in throwing found that sex-based differences "only arose from age 20 years onwards and that in individuals with throwing practice, performance disparities leveled out." Dena Crozier et al., *Gender Differences in Throwing Revisited: Sensorimotor Coordination in a Virtual Ball Aiming Task*, 13 Frontiers Hum. Neurosci. 231 (2019).

60. Given the far greater social encouragement and skills training provided to boys than to girls, it is not surprising, as Dr. Carlson notes (Carlson Rebuttal Decl. ¶ 21), that boys have the highest-ranking performances in USA Track & Field. Contrary to Dr. Carlson's suggestion that our society promotes "equal opportunities for boys and girls to participate," the reality is much different. Across the board, girls have far fewer opportunities to play sports and therefore far less coaching and skill training than boys in every age group. *See* U.S. Dep't Health & Hum. Servs., *The National Youth Sports Strategy*, 35–37 (2019), https://health.gov/sites/default/files/2019-10/National_Youth_Sports_Strategy.pdf; Aspen. Inst. Project Play, *Youth Sports Facts: Participation Rates*, https://www.aspenprojectplay.org/youth-sports/facts/participation-rates. For example, during the 2018–2019 year, fifty-seven percent of high school athletics participation opportunities went to boys, with only forty-three percent going to

girls, translating into over one million more opportunities for boys than girls. Ellen J. Staurowsky et al., Women's Sports Found., *50 Years of Title IX: We're Not Done Yet*, 30 (2022), https://www.womenssportsfoundation.org/wp-content/uploads/2022/05/Title-IX-at-50-Report-FINALC-v2-.pdf.

61. Dr. Carlson acknowledges that even the highly restrictive World Rugby policy permits transgender girls who receive puberty suppressing medication to play. (Carlson Rebuttal Decl. ¶¶ 23–24.) Dr. Carlson contends that this exception is not "grounded in scientific review of relevant data," but there is no data showing that such girls have any athletic advantage over other girls, nor is there any medically reasonable basis for assuming that they do. (Carlson Rebuttal Decl. ¶ 24.)

62. Dr. Carlson's suggestion (Carlson Rebuttal Decl. ¶ 25) that puberty suppressing medication fails to suppress the heightened levels of testosterone associated with male puberty in 25 to 49 percent of cases has no medical basis. The article he cites to support that erroneous claim is about the use of testosterone suppressant by adult transgender women who went through male puberty; it has no bearing on the efficacy of puberty suppression for transgender girls, which is highly effective and prevents transgender girls from producing the elevated levels of testosterone associated with male puberty.

63. The Klaver study does not support Dr. Carlson's claim that transgender girls who received puberty suppressing medication have an athletic advantage over other girls (Carlson Rebuttal Decl. ¶¶ 31–32) for the reasons stated in paragraph 33 above. It is not appropriate to use the Klaver article to presume that transgender girls may have more lean body mass on average than other girls because, as noted above, Klaver participants started GnRHa at much older ages than modern protocols would dictate. The findings of the study are not generalizable across decades and not relevant to the question at hand.

64. For the reasons explained in paragraphs 40 through 43 above, Dr. Carlson's claim that transgender girls "are more likely to cause concussions than other competitors"

(Carlson Rebuttal Decl. ¶ 33) has no medical basis. It is particularly unwarranted for transgender girls, like the Plaintiffs in this case, who receive puberty suppressing medication and thus do not go through male puberty.

Dr. Cantor's Supplemental Declaration

65. Dr. Cantor acknowledges that his views place him at odds with the standards of care and practice guidelines developed by the World Professional Association of Transgender Health ("WPATH") and the Endocrine Society, and which have been endorsed by a long list of major medical professional associations, including the American Medical Association, the American Academy of Pediatrics, the American Psychological Association, and many others.

66. Contrary to Dr. Cantor's unsupported claims, which implausibly cast aspersions on the integrity of our nation's leading professional medical organizations, the standards of care and practice guidelines relied upon by medical and mental health professionals who specialize in the treatment of gender dysphoria in adolescents have a sound evidentiary basis. The evidence-based methodology used to generate these guidelines is described in detail in both the WPATH Standards of Care and the Practice Guidelines promulgated by the Endocrine Society and is comparable to that used to generate similar clinical practice guidelines for other medical conditions.

67. Dr. Cantor's views, which seek to cast doubt on the existence of gender identity as a facet of human identity and to advocate the use of therapeutic techniques to discourage or prevent minors from identifying as transgender, do not have a sound scientific foundation and are distinctly at odds with the overwhelming consensus of medical science, experts, and practitioners in this area.

68. Dr. Cantor does not diagnose or treat gender dysphoria in adolescents or adults and has no training or expertise in transgender mental health care or medicine. As such, his strong disagreement with the consensus of medical experts in this area appears to be based more on his personal opinions than on a scientific foundation.

I declare under criminal penalty under the laws of Arizona that the foregoing is true and correct. Signed on the 6th day of July, 2023, in Ann Arbor, Michigan.

Daniel Shumer, M.D.

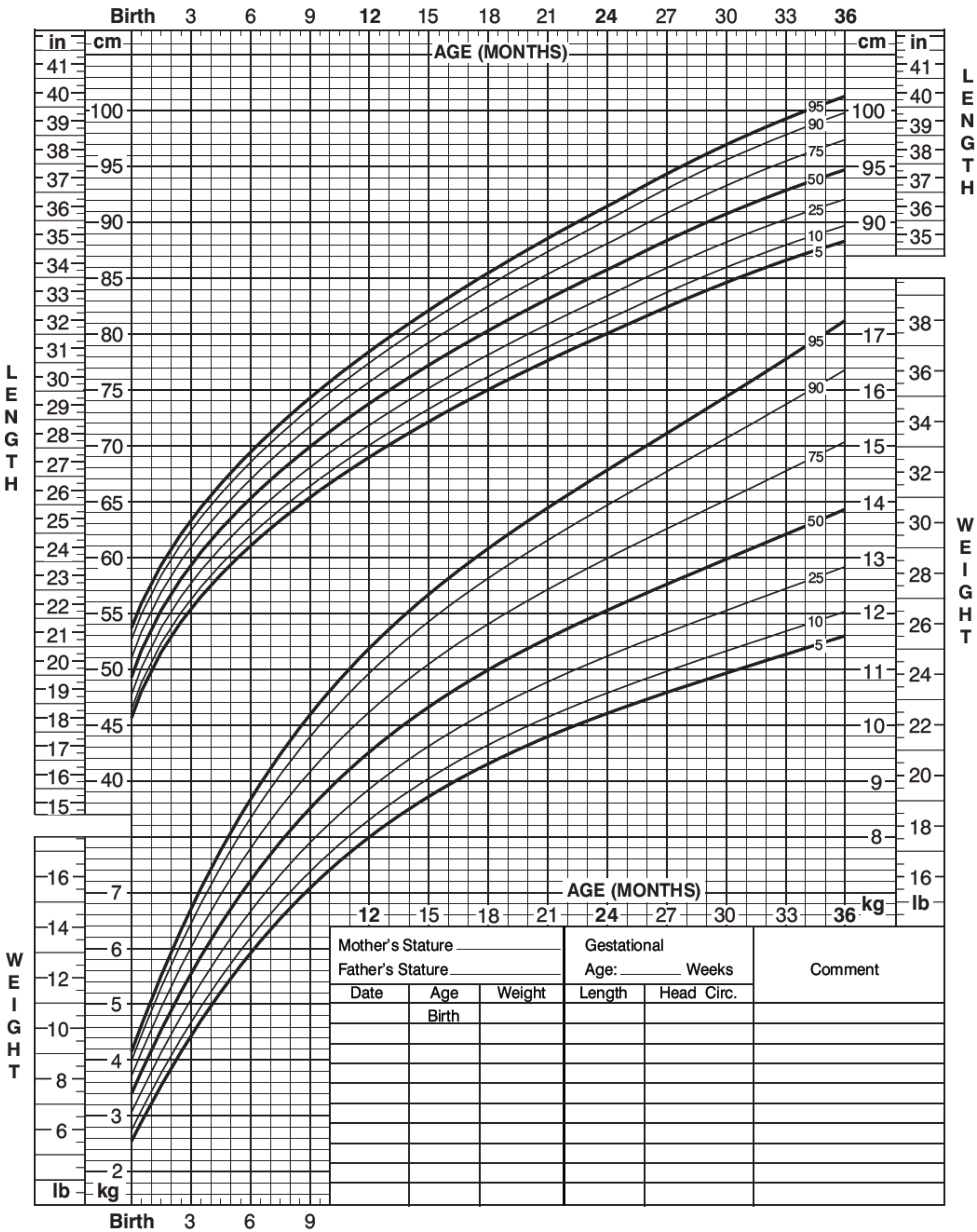
Exhibit A

CDC Growth Charts

Birth to 36 months: Girls
Length-for-age and Weight-for-age percentiles

NAME ______________________

RECORD # ____________



Mother's Stature ______			Gestational		Comment
Father's Stature ______			Age: ______ Weeks		
Date	Age	Weight	Length	Head Circ.	
	Birth				

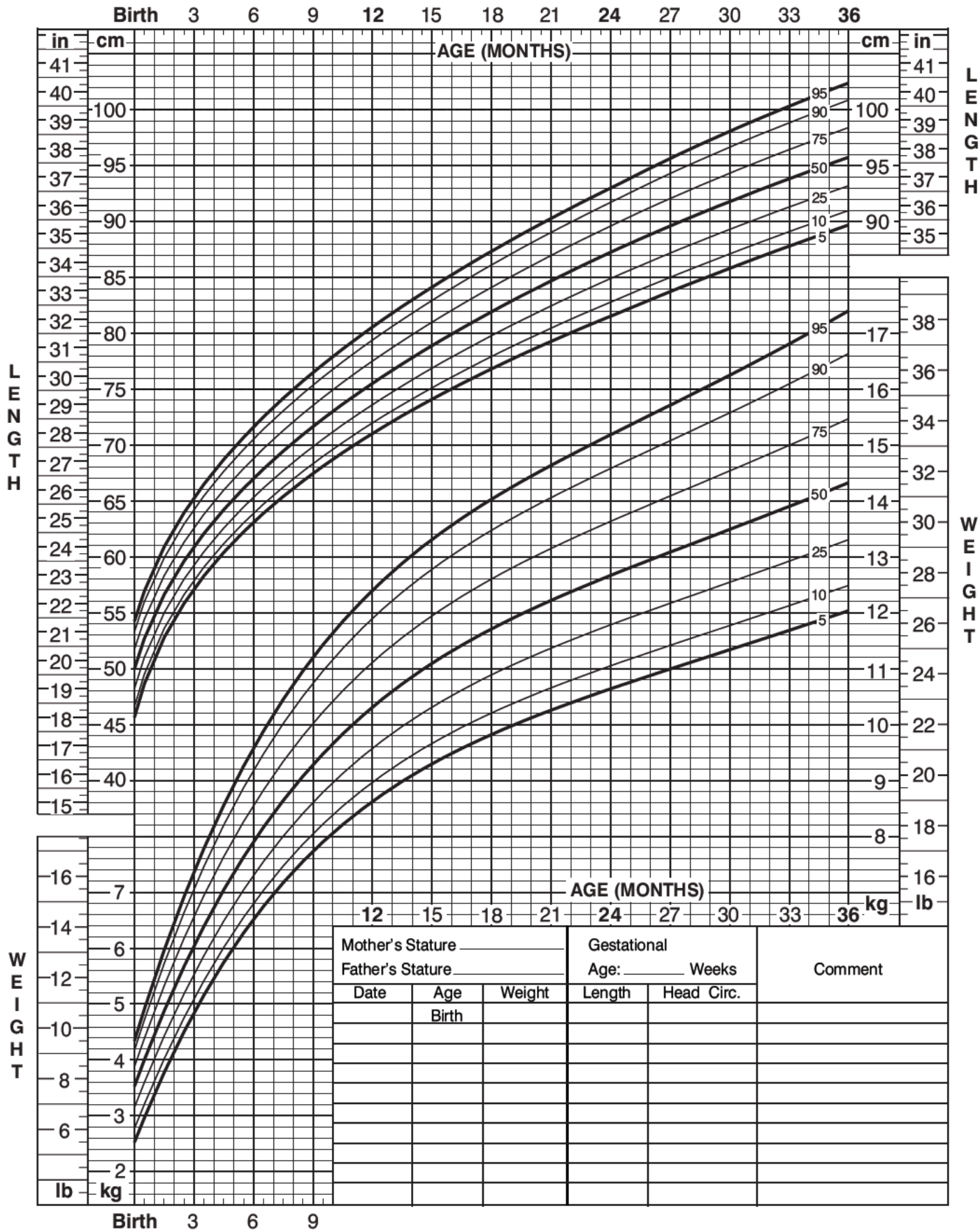
Published May 30, 2000 (modified 4/20/01).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

CDC
SAFER · HEALTHIER · PEOPLE™

Birth to 36 months: Boys
Length-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____



Mother's Stature _____ Father's Stature _____			Gestational Age: _____ Weeks		Comment
Date	Age	Weight	Length	Head Circ.	
	Birth				

Published May 30, 2000 (modified 4/20/01).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

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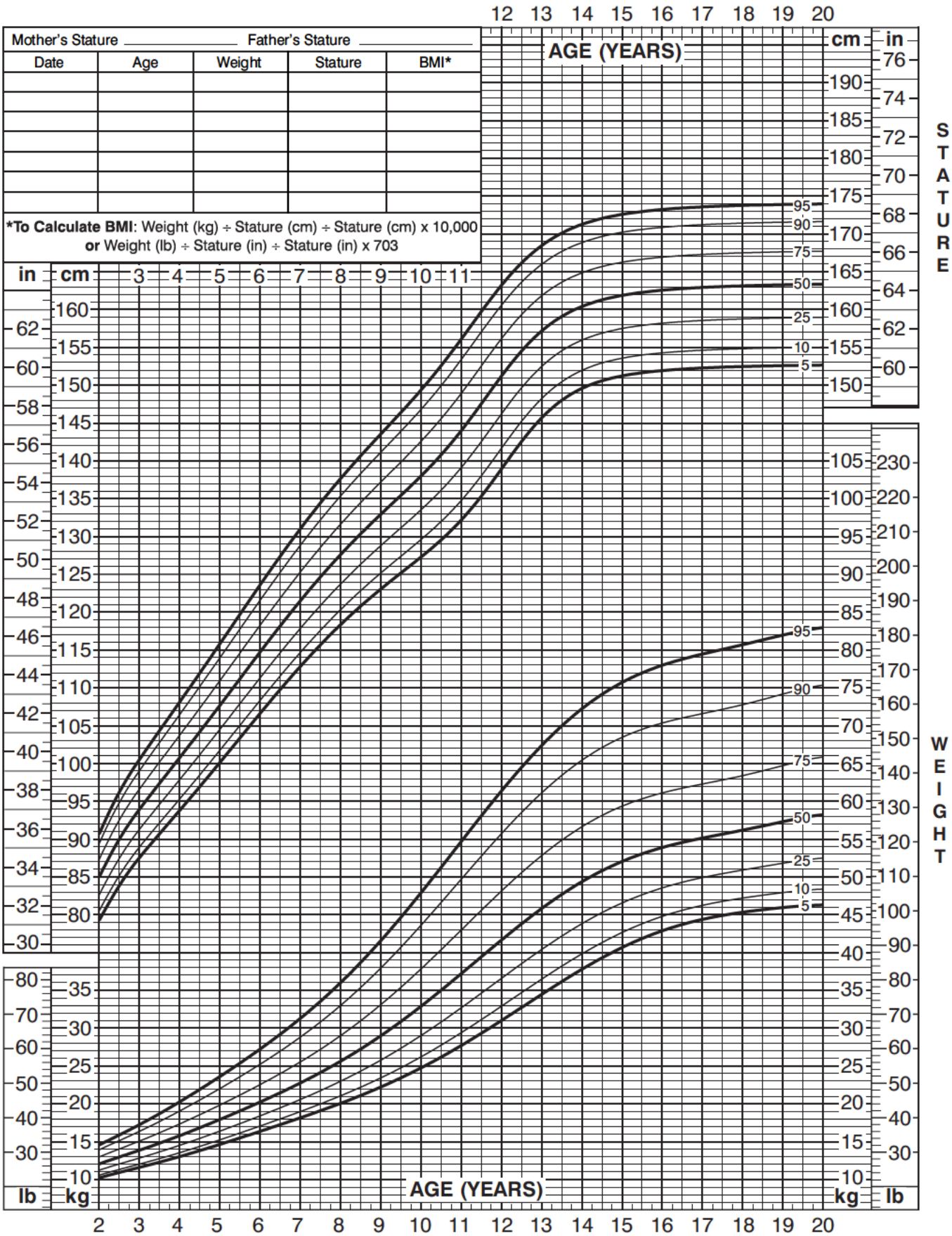
2 to 20 years: Girls
Stature-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____

Mother's Stature _____		Father's Stature _____		
Date	Age	Weight	Stature	BMI*

*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000 or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 11/21/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

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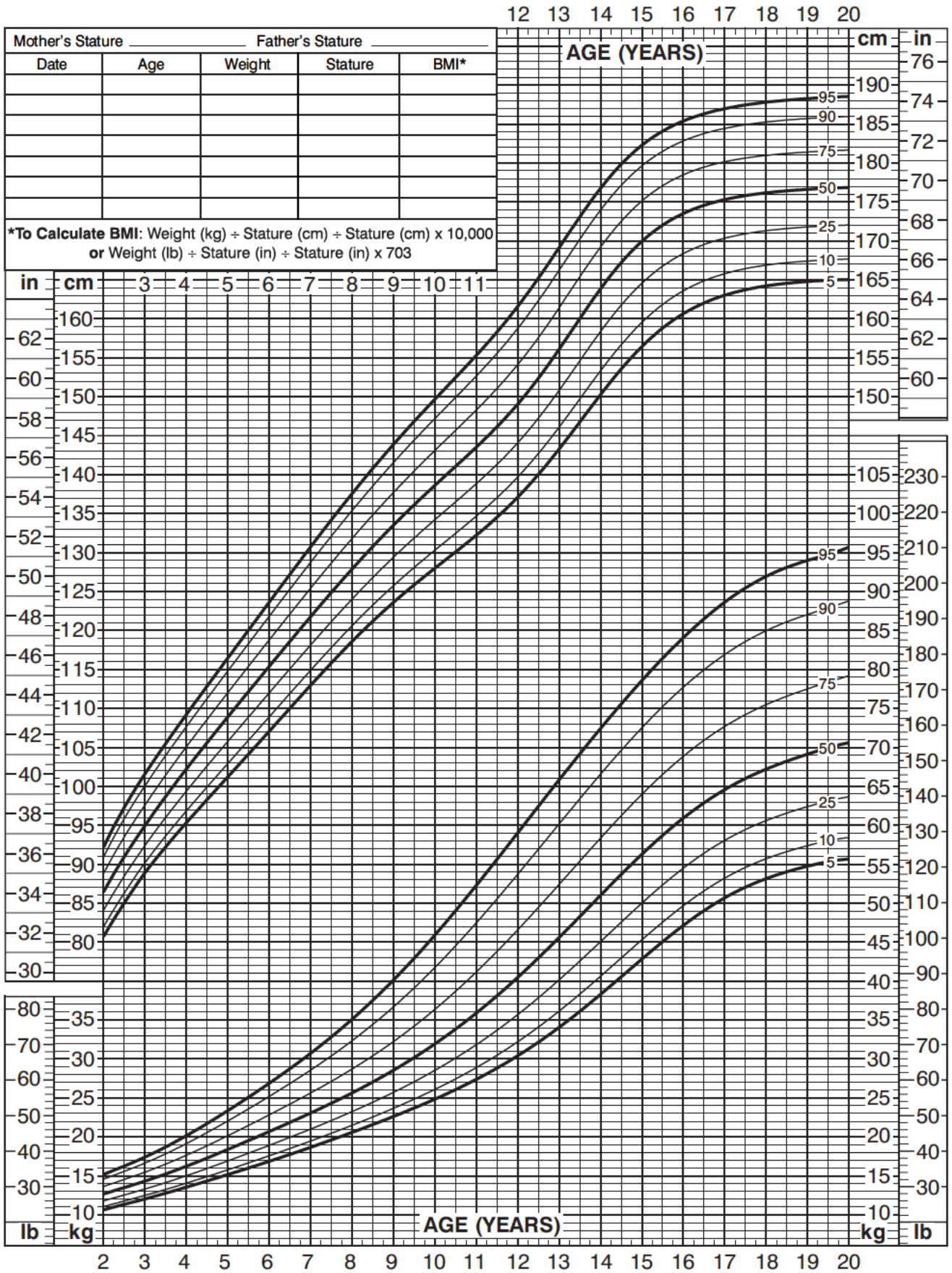
2 to 20 years: Boys
Stature-for-age and Weight-for-age percentiles

NAME _____

RECORD # _____

Mother's Stature _____		Father's Stature _____		
Date	Age	Weight	Stature	BMI*

*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000 or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 11/21/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

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Dated: August 1, 2023

Respectfully Submitted,

James Otis Law Group, LLC

/s/ D. John Sauer
D. John Sauer
Justin D. Smith
13321 N. Outer Forty Road, Suite 300
St. Louis, MO 63017
(314) 562-0031
john.sauer@james-otis.com
Attorneys for Intervenor-Defendants-Appellants

CERTIFICATE OF SERVICE

I hereby certify that, on August 1, 2023, I caused a true and correct copy of the foregoing to be filed by the Court's electronic filing system, to be served by operation of the Court's electronic filing system on counsel for all parties who have entered in the case.

/s/ D. John Sauer